

**PNEUMOTHORAX TREATMENT
OF
PULMONARY TUBERCULOSIS**
CLIVE RIVIERE

**OXFORD MEDICAL
PUBLICATIONS**

CORNELL UNIVERSITY.

THE

Roswell P. Flower Library

THE GIFT OF

ROSWELL P. FLOWER

FOR THE USE OF

THE N. Y. STATE VETERINARY COLLEGE.

1897

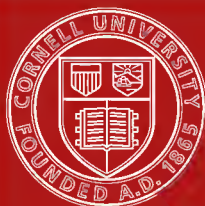
Cornell University Library
RC 311.3.P4R62

The pneumothorax treatment of pulmonary



3 1924 000 243 596

vet



Cornell University
Library

The original of this book is in
the Cornell University Library.

There are no known copyright restrictions in
the United States on the use of the text.

THE
PNEUMOTHORAX TREATMENT
OF PULMONARY TUBERCULOSIS

*Published by the Joint Committee of Henry Frowde
and Hodder & Stoughton, at the Oxford Press
Warehouse, Faleon Square, London, E.C.*

THE PNEUMOTHORAX TREATMENT OF PULMONARY TUBERCULOSIS

BY

CLIVE RIVIERE, M.D. LOND., F.R.C.P.

PHYSICIAN, CITY OF LONDON HOSPITAL FOR DISEASES
OF THE CHEST, VICTORIA PARK, E.; PHYSICIAN, EAST
LONDON HOSPITAL FOR CHILDREN, SHADWELL, E.

LONDON

HENRY FROWDE HODDER & STOUGHTON
OXFORD UNIVERSITY PRESS WARWICK SQUARE, E.C.

1917

PRINTED IN GREAT BRITAIN
BY HAZELL, WATSON AND VINEY, LD.,
LONDGN AND AYLESBURY.

PREFACE

THE treatment of pulmonary tubercle by collapse and compression of the diseased lung, through the medium of an artificial or induced pneumothorax, is a procedure which has certainly reached the stage where a convenient textbook should be welcomed. When the writer, some years since, first began to practise this treatment, he found himself greatly hampered by the difficulty of solving the various problems that confronted him through reference to a very voluminous, but scattered and highly polyglot literature. These difficulties have become no less of recent years: a convenient text-book has not arrived, and the writer has, consequently, been tempted to assemble together these results of his own and others' experience in the hope that they may smooth the way for those who wish to study this treatment.

The problems of pneumothorax treatment are many and complex, as might, indeed, be expected in a method of comparatively recent introduction, and one which involves so momentous a disturbance of the normal physical relationship of the chest organs. To some of these problems no final answer can be given at the present time, and the reader will have to content himself with an outline of recent views together with some indication of their trend. For the explanation of many phenomena the writer has been obliged to draw upon his own unsupported reasoning or experience, since the

literature, though abundant, does not touch in detail on all conceivable eventualities. Nevertheless the references to authority in the text are necessarily copious—so much so, indeed, as to have demanded, in the interests of practical utility, a somewhat severe pruning when the bibliography was reached. Into this latter have been admitted only such references as seemed to the writer of real historical interest, of conspicuous clinical value, or containing original work or first-hand information on points of special importance. To this limited degree the book may serve as a work of reference—the reader will obtain introduction to some of the most notable articles in the literature.

Pneumothorax treatment can be regarded as no less than a great and beneficent discovery; if this small volume helps to promote its progress or reputation in any degree, the writer will feel that his labour has not been expended in vain.

QUEEN ANNE STREET,
LONDON, W.

CONTENTS

	PAGE
INTRODUCTION	1

CHAPTER I

THE MODE OF ACTION OF ARTIFICIAL PNEUMOTHORAX

MECHANICAL EFFECTS	6
SEROLOGICAL EFFECTS	8

CHAPTER II

INDICATIONS AND CONTRAINDICATIONS

CLINICALLY UNILATERAL TUBERCULOSIS	12
APICAL PHTHISIS	16
The Better Lung	17
Early Phthisis	19
ACUTE FORMS OF TUBERCULOSIS	20
HÆMOPTYSIS	21
SPECIAL CONTRAINDICATIONS	22
Lungs	22
Circulatory Organs and Kidneys	22
Laryngeal Tuberculosis	22
Intestinal Tuberculosis	23
Diabetes	23

CHAPTER III

PNEUMOTHORAX APPARATUS

	PAGE
GENERAL FEATURES	24
THE MANOMETER	28
THE GAS TO USE	31
TUBING	33
NEEDLES	34
PREPARATION OF APPARATUS FOR USE	37

CHAPTER IV

TECHNIQUE OF INITIAL OPERATION

NURSING HOME OR PRIVATE HOUSE	39
PRELIMINARIES	39
SELECTION OF SITES FOR PUNCTURE	40
THE OPERATION	42
Preparation of Patient	42
Preparation of Instruments and Apparatus	42
Position of Patient	43
Passing the Needle	43
Manometric Response	45
Entrance of Gas	45
Amount of Gas at first Operation	46
Manometer Readings	47
Removing the Needle	47
Effect on Chest Signs	48
Hints for First Operation	48
DIFFICULTIES ATTENDING INITIAL OPERATION	48
Absence of Manometric Response	49
Incomplete Manometric Response	50
Negative Pressure without Respiratory Fluctuations	50
Pleural Pockets	50
Puncture of the Abdominal Cavity	51

CHAPTER V

EFFECT OF PLEURAL ADHESIONS: PARTIAL
AND COMPLETE PNEUMOTHORAX

	PAGE
PARTIAL PNEUMOTHORAX	53
Ineffectual, with Example	53
Effective, with Example	55
COMPLETE PNEUMOTHORAX, with Example	56
DIAGNOSIS OF ADHERENT PLEURA	60
REDUCTION OF ADHERENT PLEURA	61

CHAPTER VI

REFILLS

INTERVALS AT EARLY STAGE	64
OPERATION OF REFILLING	64
MANOMETER READINGS	66
Intrapleural Pressure	66
Respiratory Fluctuations	69
Re-expansion of Lung	70
Condition of Mediastinum	70
PUTTING IN GAS	71
EARLY FILLINGS, QUANTITY AND PRESSURE OF GAS	71
COMPLETE COLLAPSE	71
Its diagnosis from Radioscopy	72
" " " Physical Signs	73
" " " Symptoms	73
INTERVALS AT LATER STAGES	74
INTRAPLEURAL PRESSURES	77
Complete Pneumothorax	77
Partial Pneumothorax	78
Effect of Altitude on Gas Pressure	80

CHAPTER VII

	PAGE
PHYSICAL SIGNS IN ARTIFICIAL PNEUMOTHORAX	82

CHAPTER VIII

GENERAL RÉGIME: THE COURSE OF TREATMENT AND ITS TERMINATION

DISAPPEARANCE OF SYMPTOMS	88
Temperature and Pulse	89
Cough and Expectoration	90
Weight	92
CLINICAL RECOVERY	92
ANATOMICAL RECOVERY	94
TERMINATION OF TREATMENT	97
Partial Pneumothorax	97
Complete Pneumothorax	97
RE-EXPANSION OF THE LUNG	98

CHAPTER IX

PNEUMOTHORAX AS PALLIATIVE TREATMENT	101
--	-----

CHAPTER X

EFFECTS OF PNEUMOTHORAX ON THE THORACIC ORGANS

THE MEDIASTINUM	104
Open Pneumothorax	104
Closed Pneumothorax	105
THE DIAPHRAGM	106

CONTENTS

xi

PAGE

THE COMPRESSED LUNG	108
THE FUNCTIONING LUNG	111
THE LUNG CIRCULATION	114
HEART AND CIRCULATION	116

CHAPTER XI

PLEURISY AS A COMPLICATION

PREVALENCE	120
CAUSATION	121
DRY PLEURISY	123
PLEURISY WITH EFFUSION	123
General Classification	123
Purulent Effusions	125
CHEMISTRY OF EFFUSIONS	127
CYTOLOGY	129
BACTERIOLOGY	129
MAYER'S CLASSIFICATION	130
(1) Exudates due to Tuberculous Pleuritis	130
(2) Exudates of Acute Infectious Nature	130
(3) Exudates due to Perforation of Visceral Pleura	130
(4) Exudates of a Special Type: the com- monest Form	130
EFFECTS OF PLEURISY	131
Beneficial Effects	131
Unfavourable Effects	133
SYMPTOMS	134
EFFECTS OF EFFUSION ON MANOMETER READINGS	136
PHYSICAL SIGNS	138
Dry Pleurisy	138
Effusions	139

	PAGE
TREATMENT	140
Chronic Effusions	141
Acute Serous Pleurisy	142
Acute Purulent Effusions	143

CHAPTER XII

GAS REPLACEMENT

INSTRUMENTS NEEDED	145
THE OPERATION	145

CHAPTER XIII

THE ACCIDENTS OF PNEUMOTHORAX
TREATMENT

PLEURAL SHOCK	151
Symptoms	151
Conditions of Appearance	152
Prophylaxis	154
Treatment	155
GAS EMBOLISM	156
Mechanism of Gas Embolism	156
Intravenous Pressure	157
Source of Gas Embolism	158
Alveolar Air	158
From the Manometer Tubing	158
Volume and Nature of Gas	159
Symptoms	160
Prophylaxis	160
Treatment	161
SURGICAL EMPHYSEMA	161
Superficial	161
Deep or Mediastinal	162

CONTENTS

xiii

	PAGE
PERFORATION OF THE LUNG	162
Symptoms	163
Prognosis	163
Prophylaxis	164
Treatment	164

CHAPTER XIV

GENERAL RESULTS OF TREATMENT .	166
BIBLIOGRAPHY	175
INDEX	181

LIST OF ILLUSTRATIONS

FIG.	PAGE
1. PEARSON AND LILLINGSTON PNEUMOTHORAX APPARATUS	26
2. WRITER'S TROCAR AND CANNULA	34
3. LILLINGSTON'S NEEDLE	36
4. TEMPERATURE CHART SHOWING LOSS OF FEVER . . .	58
5. PARAVERTEBRAL TRIANGLES AND QUADRANGULAR SURRESONANCE	85
6. AUTOINOCULATION FOLLOWING REFILLS IN PARTIAL PNEUMOTHORAX	88
7. AUTOINOCULATION FOLLOWING REFILLS WITH BILATERAL DISEASE	89
8. TEMPERATURE CHART SHOWING LOSS OF FEVER . . .	90
9. TEMPERATURE CHART SHOWING DELAYED DEFERVES- CENCE DUE TO INCOMPLETE COLLAPSE	91
10. TYPES OF FEVER ASSOCIATED WITH PURULENT EFFU- SIONS	126

PLATE	FACING PAGE
I. RADIOGRAM OF CHEST SHOWING PARTIAL PNEUMO- THORAX	54
II. RADIOGRAM OF CHEST SHOWING COMPLETE PNEUMO- THORAX	59

THE PNEUMOTHORAX TREATMENT OF PULMONARY TUBERCULOSIS

INTRODUCTION

No more hopeful ray of sunshine has ever come to illumine the dark kingdoms of disease than that introduced into the path of the consumptive through the discovery of artificial pneumothorax. Recommended on theoretical grounds nearly a century ago, so soon, indeed, as the elasticity of the lung was first clearly recognised, first practised only some sixty years later, but again forgotten, it is only during the present decade that it is reaching that place of supreme importance in phthisis-therapy which is undoubtedly its due. In 1821 James Carson of Liverpool, in a series of enlightened physiological essays, first urged its trial in the treatment of disease. He pointed out in the clearest and most convincing manner how certain were the advantages to be derived from pulmonary collapse both in phthisis and in other diseases, and proved its feasibility by animal experiment. His views and some quotations from his work will be set forth more fully in a succeeding section (pp. 6 and 7). Nothing came of these recommendations at the time, which was, perhaps, just as well in those pre-antiseptic days. True, a certain Dr. F. H. Ramadge is credited with having punctured the chest wall somewhere about the year 1832, but the writer can find no evidence of this reputed operation. Nevertheless in this year (1832) James Houghton was reporting improvement in a case of advanced phthisis as the result of a

spontaneous pneumothorax—the patient, a bricklayer, was found thereafter working twelve hours a day at building a chapel. A few years later, in 1837, Stokes was also pointing out the decided improvement which spontaneous pneumothorax might sometimes exert on the course of phthisis. “Of the temporary suspension of the symptoms of phthisis in cases of perforation,” he said, “there have been now many examples. If the perforation has occurred in a case where tubercle was not yet deposited in great quantity, and above all, where it had not yet appeared in the opposite lung, the accident in question really produces a temporary cure of consumption.” It was not, however, till the 'eighties that the subject of pneumothorax again aroused special interest, to find then, at last, practical realisation.

Toussaint, a pupil of Potain's, in the year 1880 once again set forth on clinical grounds the claims of pulmonary collapse, to be followed in 1882 by Forlanini, who, however, failed to put the matter to the practical test till the year 1888. Meanwhile Potain in 1884 actually treated a case of spontaneous hydro-pneumothorax by replacing the fluid with sterilised air on repeated occasions, “une méthode non usitée jusqu'ici.” He further treated two other patients by the same method, reporting the three cases in the year 1888. All recovered, though one suffered a fatal relapse at a later date. He used a manometer in these operations, and advised renewing the air in certain cases of spontaneous pneumothorax so as to leave the intrathoracic pressure somewhere between 7 mm. Hg. and atmospheric pressure. Later he affirmed the superiority of nitrogen as the most inert gas, and that least rapidly absorbed. At about the same time and independently, in 1885, Cayley treated a case of severe hæmoptysis in phthisis by open incision of the chest wall, and reported it to the Clinical Society of London. The hæmorrhage was largely arrested, though adhesions prevented complete collapse, and the patient died five days later with miliary tuberculosis.

From this time onwards the matter appeared again to drop out of notice, though Forlanini was treating some cases, and reported them in 1894 and 1895. In 1898 J. B. Murphy of Chicago was urging the treatment on grounds of surgical principle, and himself treated five cases, his operation being carried out with a trocar and cannula. His pupil Lemke a few years later (1901) reported the treatment of fifty-three phthisis cases, many of them with fairly early disease, by a similar method. In the interval Schell of Indiana, in 1899, had described a case in which an artificial pneumothorax was resorted to for the cure of hæmoptysis. These publications attracted the notice of Brauer, to whom this method of treatment has since been indebted for much profitable speculation and research. His method of operation, by open incision down to the parietal pleura and perforation of this with a blunt cannula, has since been practically abandoned, but the safety achieved by the introduction of the water-manometer still stands to his credit. His first cases were reported in 1905 and 1906; Adolph Schmidt also reported a case in the latter year. Meanwhile Forlanini had been working in silence, and in the year 1906 was able to report twenty-five cases of pulmonary tuberculosis submitted to treatment since his earlier publication.

Since then pneumothorax treatment has found its way into all countries where the medical sciences can be said to flourish. In Italy Forlanini has attracted a distinguished following; the method has returned to France with F. Dumarest (1908) and Dessirier (1908), with Balvay and Areelin (1909), and with Küss (1910), and P. Courmont (1910); to America with Harris, Mary Lapham (1911) and Robinson and Floyd (1912); to England with Colebrook, Lillingston, Vere Pearson and Rhodes, all in 1911, though before that time, in 1905, Stuart Tidey was extolling its principles at the International Tuberculosis Congress in Paris. In Germany, Brauer was followed by Adolph Schmidt (1906) and Lexer (1906), and by Klemperer, Wellman and

others. Meanwhile Denmark had been adding distinguished share to pneumothorax treatment through Saugman (1907) and Begtrup-Hansen; and Switzerland through L. Spengler, v. Muralt and a host of others.

Thus the treatment of phthisis by artificial pneumothorax, first suggested on theoretical grounds, later supported on clinical grounds derived from the observation of cases of spontaneous pneumothorax, now at last holds a firm place on grounds of its own undoubted practical value. Of no method of treatment, perhaps, does less difference of opinion exist as to application, technique, and, above all, utility among those who have had fair opportunity for its study. "All the physicians who have applied artificial pneumothorax among a notable number of patients," remarks a French writer, "agree in assigning to it an eminent therapeutic value. We have not seen, up to now, an equal unanimity in the appreciation of any antituberculous remedy, and this agreement alone is of a kind to give us encouragement." But in another important particular also pneumothorax treatment may be claimed to differ very markedly from all other remedies used in the treatment of tuberculosis—it is especially applicable to the treatment of advanced disease. "It is a noteworthy fact," writes a distinguished physician, "that practically all the methods of treatment and remedies which have been recommended during the past three or four decades for the relief and cure of phthisis, have been stated to exercise their alleged curative effects only during the incipient stage of the disease, when diagnosis is doubtful in a large proportion of cases, and spontaneous cure is not unknown. It is therefore refreshing to find that the newest method of treatment of pulmonary tuberculosis is recommended for far advanced cases, especially those manifesting a tendency to pursue a rapidly fatal course, and showing signs of extensive excavations. When every other method for checking the progress of the process in the lung has been tried and found wanting; when fever consumes the body, or life is threatened with

copious and uncontrollable pulmonary hæmorrhages, this method of treatment may be applied with striking success." This, indeed, is the rôle of artificial pneumothorax, to rescue those formerly beyond the bounds of human aid, to restore the lost to life, or at least to a measure of comfortable existence. The indications for its use and also the contraindications, the measure of its achievements and their limitations, and the most important of those points on which success or failure may turn, will be duly set forth in the following pages.

CHAPTER I

THE MODE OF ACTION OF ARTIFICIAL PNEUMOTHORAX

THOUGH a study of the effects of collapse of the lung in pulmonary tuberculosis must be expected to involve many obscure theoretical problems, yet to the clinician two clearly defined factors stand out for main consideration—on the one hand the local mechanical effects, and on the other the removal of toxæmia.

MECHANICAL EFFECTS

These and their possible application to the treatment of disease were noted so soon as the elastic recoil of the lungs was first a recognised fact; James Carson, as already remarked, was urging the production of artificial pneumothorax as early as 1821. He pointed out how great a bar to healing processes were the elasticity, condition of extension, and constant movement of the lungs. In abscess of the lung he remarked “the sides of the abscess are prevented from falling into a salutary contact, not by the matter which lodges between them, but by the powerful elasticity and retraction of the surrounding substance.” As soon, he says, expect healing in a divided tendo Achillis without mechanical aid. By collapse of the lung, on the other hand, “the diseased part would be placed in a quiescent state, receiving little or no disturbance from the movements of respiration which would be performed solely by the other lung, and the divided surfaces would be brought

into close contact by the same resilient power which before had kept them asunder. A wound or abscess in this lung would be placed in circumstances at least as favourable to the healing process as the same affections on any other part of the body." Finally he recommended its application to the treatment of "consumption"—"in those cases in which the disease is placed in one of the lungs only, the remedy would appear to be simple, safe, and complete." Further on he remarks that "a hæmorrhage from one of the lungs, the frequent prelude of consumption, if not immediately fatal, would be as certainly stopped by the collapse of that lung, as the flooding consequent upon parturition by the contraction or rather the resilience of the womb." He was by no means blind to the danger, by sudden collapse, of flooding the tubes with blood or pus, and advised reducing the lung "to a state of collapse by degrees only." "It has long been my opinion," he says, "that if ever this disease (phthisis) is to be cured, and it is an event of which I am by no means disposed to despair, it must be accomplished by mechanical means, or in other words by a surgical operation."

This sound statement of the manifest mechanical advantages to be expected from pulmonary collapse can hardly be bettered by any quotation from later work. But to it must be added certain less obvious factors, still in the main mechanical, which must also minister to the beneficial results, though in a degree which is at present uncertain and even contentious. These are, primarily, the effect on the growth of the tubercle bacillus of the diminution of oxygen in the collapsed lung, and of the rapid growth of fibrous tissue; and, secondarily, the effect on this latter process of circulatory changes, whether hyperæmia or its opposite, of lymph stasis, and of the imprisonment of toxins. Each of these has been expanded into the rôle of an explanatory hypothesis in the hands of one or more authorities, though it has seemed to the writer more reasonable to regard them as but notable factors in the

total result. The local improvement foreshadowed by Carson has generally been well attained in practice. Under artificial pneumothorax, as will be more fully described elsewhere, the lung is retracted to a firm, airless, fleshy mass, cavities and tubes are flattened out, their surfaces become adherent, and the diseased parts, and in some cases healthy parts also, become permeated with a dense growth of fibrous tissue. In such a lung, tuberculous processes are always in abeyance, and no fresh tubercles develop, a fact which post-mortem evidence has clearly established. So much for the local facts associated with pulmonary collapse—one side of the picture and certainly a very important one.

SEROLOGICAL EFFECTS

The other side of the picture shows itself in the removal of toxæmia as exemplified by loss of symptoms, fever, sweats, malaise, wasting, and therewith improvement of general health. It can hardly be doubted that these effects, analogous to those produced through reduction of autoinoculation by other means, are due to some mechanical bar to the absorption of toxins. In the collapsed lung fluid excretions are evacuated, and can no more accumulate, caseous areas are rendered drier, cavity walls unite, and the diseased surface is, at worst, enormously reduced in area. In addition a stasis of lymph is brought about by immobilisation, the lymph channels are distended as was shown by Bruns, Warnecke, and Kistler, and absorption reduced to a minimum. The blood supply may be large or small according to the intrapleural pressure, and since toxic absorption does not in practice vary with this pressure, the removal of toxæmia, which is so important a feature of pneumothorax treatment, may well be ascribed, in the main, to lymph stasis.

In what way, it may be asked, does the removal of toxæmia or autoinoculation act? Not only does it release the nervous system and all the organs from a

poisoning which has seriously interfered with health, but it at once breaks through that "vicious circle" which has favoured the spread of disease in the other lung and elsewhere. For, as in overdosage with tuberculin so in autotoxæmia, every large "general reaction" exhibited in fever, malaise, and other symptoms is accompanied by that stirring up of all the tuberculous foci known as a "focal reaction." In this way the active foci are flooded with blood, a further supply of toxin is carried into the circulation, and further "reactions" result; in addition these foci are helped to extend and grow, quiescent disease becomes active, and new centres arise. The removal of tuberculo-toxæmia curtails those focal reactions in the better lung which were leading to the spread of disease, and thus helps to bring about in that organ the improvement which almost invariably follows. It may well be, also, that, as many believe, a favourable hyperæmia of this lung may occur and assist the healing process (pp. 115 and 116). In association with the removal of excessive reactions the opsonic index rises as would be expected, and as has been demonstrated by Pigger, working under L. Spengler, by Carpi, and others.

The removal of toxæmia is so important a feature in pneumothorax treatment that the writer is always less inclined to recommend the operation in cases where definite toxæmia is absent, and where, in consequence, the mechanical effects can alone be brought into play. Where, however, large cavities can be compressed and widespread disease obliterated, where, in short, the mere character and extent of disease render healing well-nigh impossible in an extended lung, the mechanical advantages belonging to collapse are not to be withheld. But their influence by itself on the growth and development of tubercles, at least under experimental conditions, appears to be somewhat in doubt. Breccia, indeed, finds that pulmonary collapse in animals will prevent the spread of disease to this lung; but Schur and Plaschke observed the development of tuberculosis in

the compressed as well as in the functioning lung of animals submitted to tubercle inoculation, whether intravenous or intratracheal. On these grounds they argued that the removal of toxæmia was the all-important factor in pneumothorax therapy, and not the pulmonary collapse.

Other serological influences may well be at work in cases of pneumothorax therapy, perhaps pleural influences analogous to those which often lead to general improvement as the result of a pleurisy, even a dry one, in cases of phthisis. These, if they occur in pneumothorax, are apparently enhanced in certain cases by the development of a pleural effusion, but of their nature we have, at present, no certain knowledge.

CHAPTER II

INDICATIONS AND CONTRAINDICATIONS

THE success of pneumothorax treatment is not a little bound up in the suitable selection of cases. Indicated for progressing, marked, or even advanced disease, it is, nevertheless, only applicable to a selected proportion of such material, although the majority of advanced cases must at some time or other have passed through a stage where a useful pneumothorax was at least a possibility. Among a mixed phthisis material containing patients at all stages of the disease, the incidence of cases immediately suitable for pneumothorax therapy appears to be a rather small one. Keller puts it at 7% with only 3.9% successfully achieved. Ferrio finds 5 or 6% fully suitable among 700 cases examined, but thinks that this number could be increased with experience. Fishberg notes 22 suitable cases among 210, or 10%, but regards this figure as high because he was at the time agitating to obtain such cases; he believes the usual incidence to be below 5%. Bernard found 22 among 628 cases or 3.5% suitable, Courmont 31 of 352 or 9%. These percentages of suitable cases, however, based on a mixed material, necessarily take no heed of past or future—ignore, in other words, the fact that most progressive phthisis cases at some time in their course must reach a stage when pneumothorax treatment might be used with advantage, and that a case unsuitable to-day might have been suitable yesterday, or may become so to-morrow. This fact should be taken into account in gauging the proportion of phthisis cases suitable for

pneumothorax treatment, particularly since it is one which must gain increasing importance as the value of the method becomes more and more widely known. For the physician well acquainted with the method and familiar with its indications and contraindications will not allow his patients to pass beyond the point at which this, and with it every other form of treatment, becomes too late, but will seize the suitable moment in each case, and thus ensure that the benefits of pneumothorax treatment are extended to a much larger proportion of phthisis material than could be the case at the present time.

What may be called the "classical" case for artificial pneumothorax is one of severe one-sided disease with the other lung clear or nearly clear to physical examination. These cases, less uncommon than might at first sight be expected, must form a large proportion of any well-selected pneumothorax material; they appear to represent a distinct group capable of being separated off from more ordinary adult cases of pulmonary tuberculosis. For ordinary apical phthisis of adults spreads but a little way in one lung before it presents physical signs at the opposite apex, and the conditions of advanced one-sided disease can thus never be reached. The unilateral case, so suitable both in theory and in practice for pneumothorax therapy, stands in a group apart, and merits special attention in a book devoted to this form of treatment.

CLINICALLY UNILATERAL TUBERCULOSIS

Since the writer's hospital pneumothorax material has been mainly composed of advanced one-sided cases, a reference to these will serve conveniently to illustrate the peculiarities of the group under consideration. Among twenty of these cases, all adults, where the type of disease could be decided with some probability, seventeen appeared to stand in a group by themselves, presenting points of difference from ordinary apical phthisis. All

of these showed disease covering the whole of one side, and in all the good lung was practically clear of physical signs. In six no signs of cavitation were present, but large moist crepitations extended from apex to base, in two on the right and in four on the left side. In eleven cavitation was evident to physical examination, in most of considerable extent, but in all it presented one striking peculiarity. In no case was it in proximity to the apex; in all, the cavities were found comparatively low down in the lung. Among four with right-sided disease, in three cavitation was in the axillary region, and in the fourth at the base. In the remaining seven disease was on the left side and in six of these cavitation was just outside the heart about the middle of the lung, or in the axilla, or close above the heart; in one it was basal. In practically all the cases notable evidence of fibrosis with displacement of heart and mediastinum was present, particularly, as would be expected, in the cases with cavitation. The good lung, as has been mentioned, was clear to stethoscopic signs in all these cases, save for doubtful sounds which proved, in the light of subsequent events, to have been conducted from the diseased side.

The use of the X-rays, however, soon dispelled the illusion that this better lung was in reality free of disease. In all cases there was visible a definite peri-bronchial tuberculosis, in most cases indeed quiet or arrested, spreading out from the hilum and often reaching a considerable way towards the surface. This occurrence of deep disease without surface signs is immediately reminiscent of hilum tuberculosis of children, and further comparison brings conviction that these clinically unilateral cases are none other than a parallel condition, hilum tuberculosis, in the adult. The main characteristic of this type of disease appears to be its tendency to a chronic course with little or no symptoms until a late stage is reached. Then at last toxæmia appears sufficient to overcome tolerance and the symptoms of an active autotoxic tuberculosis arise. By this time disease may have reached the surface on one side, and physical signs

appear almost simultaneously over the whole or nearly the whole lung. On the other side, no physical signs of disease may be present, and the impression of a truly one-sided process is thus wrongly given since disease may, in reality, involve the whole of this lung except the immediate surface. If disease is active on this side also, a condition which is most easily discoverable to radiographic examination, the case is not suitable for pneumothorax treatment. But often disease on this side is of a quiet type or arrested, and the appearance of such a process on the plate, even if of wide extent, need not in practice deter from the attempt at pneumothorax treatment, since the results of this often prove themselves as good as if the lung had been really healthy.

Unilateral tuberculosis as a distinct type of disease was first discussed by Straub and Otten in 1912. They described twenty-seven cases mostly in children or young people, and they recognised, moreover, its peculiar relationship to pneumothorax treatment, and included in their paper an analysis of 99 cases treated by Brauer of Hamburg after this method. The main characteristics on which they laid stress were—the low level of the oldest process and hence of cavitation; the appearance of signs first over the front of the chest, the marked tendency to fibrosis and displacement of organs, and the more chronic course of left-sided disease proceeding in many cases to cavitation without complaint of either cough, sputum, fever or pain; the characteristic spread of disease on the right side into the axilla, and on the left side towards the surface just above the heart.

They analysed some general tuberculosis material to discover the incidence of this “unilateral” type of disease. Among 135 cases of pulmonary tuberculosis, they found left-sided disease of this type, chosen as being most readily recognised, in 10 cases or 7·4%. Among Brauer’s 99 pneumothorax cases, 48 had right-sided and 51 left-sided disease. Among the latter 17 or 33·3% were certainly of this type, and a further 18 or 35·3% probably so. They produced figures also to

show that the results of pneumothorax treatment were strikingly better in this class of case than in cases of ordinary type. Their paper also contains an interesting comparison of the results of pneumothorax treatment with those of ordinary sanatorium treatment in these cases.

	Much improved.	Improved.	No change.	Died.
Brauer's 17 left-sided tuberculous cases treated by Pneumothorax . . .	$\left\{ \begin{array}{l} 10 \\ = 58.8\% \end{array} \right.$	$\left\{ \begin{array}{l} 5 \\ = 29.4\% \end{array} \right.$	—	$\left\{ \begin{array}{l} 2 \\ = 11.8\% \end{array} \right.$
Straub & Otten's 27 cases under Sanatorium treatment . . .	$\left\{ \begin{array}{l} 2 \\ = 10.5\% \end{array} \right.$	$\left\{ \begin{array}{l} 7 \\ = 36.9\% \end{array} \right.$	$\left\{ \begin{array}{l} 3 \\ = 15.7\% \end{array} \right.$	$\left\{ \begin{array}{l} 7 \\ = 36.9\% \end{array} \right.$

We thus find that among cases suitable for pneumothorax treatment, first place must be given to clinically unilateral tuberculosis, an outcome, though only an occasional one, of hilum tuberculosis in the adult. When the case is first seen severe disease is already manifest as a rule over a considerable part of one lung, though but a short history of symptoms may be obtainable. The type and extent of disease hidden in the apparently healthy side must necessarily be a matter of doubt. Dyspnoea cannot be taken as clear evidence of widespread lung involvement, since it may be of toxic origin. The slightest degree of cyanosis, on the other hand, seems to the writer of ominous significance. When disease is active and near the surface on this side, the earliest signs usually show themselves in front, in axilla and near the base. The only definite evidence, however, of the condition of this lung, is obtainable by means of a radiogram. For the purposes of pneumothorax treatment shadows pointing to arrested or quiet disease, even though this is of wide extent, may generally be ignored. Active disease is a relative contraindication unless limited to the lung immediately around the hilum,

APICAL PHTHISIS

Pulmonary tuberculosis of the ordinary apical type is suitable for pneumothorax treatment under much closer limitations. In two particulars it stands in marked contrast to the class of case, hilum tuberculosis, already described; firstly, disease cannot be allowed to spread far without risk of the opposite lung becoming decidedly involved; secondly, disease in the better lung is more likely to be active and to constitute a serious menace to successful treatment. As a matter of fact the first of these is only an apparent difference, depending on the fact that apical disease gives manifest signs at a stage where central disease is still of necessity hidden. But the tendency to a more active type of disease on the better side in apical phthisis constitutes a real distinction and one that especially governs the use of artificial pneumothorax in this disease.

Where, then, should the indications and contraindications be drawn in a case of apical phthisis? Forlanini warns us to think of pneumothorax treatment as soon as ulcerative processes appear in the lungs, and this for the following reasons:—

(a) The probability of disease remaining one-sided becomes smaller the further the process advances.

(b) The more advanced the disease the greater is the likelihood of adhesions limiting or altogether preventing this treatment.

(c) The more circumscribed the disease the more assurance is there that the lung will expand and functionate perfectly when treatment is completed.

(d) The more advanced the disease the greater is the risk of that fatal accident, perforation of the lung. These warnings against delay are applicable to hilum tuberculosis no less than to phthisis, but in that condition it commonly happens that the extent of disease has only been realised when it is already far advanced. In apical phthisis the most vital of these warnings is the first, the danger, namely, that active disease may

appear in the better lung and jeopardise or altogether prevent treatment by artificial pneumothorax. As a rule this will have happened before the other three warnings need serious consideration, so that our first and main anxiety must be the state of the better lung.

The Better Lung.—The favourable influence which pneumothorax treatment exerts on the lesions in the functioning lung is not the least notable of its achievements, but these lesions must be reasonably suitable both in quality and quantity for this benefit to accrue. Most writers are of opinion that active disease in the better lung is a distinct contraindication; inactive or quiet disease is considered no bar. These qualifications are obviously open to various interpretations—the decision must always be directed to the conditions present in the individual case. As regards the activity of lesions, Forlanini finds that limited active lesions may improve; Weiss remarks that they may sometimes be favourably influenced—he has at least seen them uninfluenced. L. Spengler, on the other hand, regards a fresh active process on the better side as an absolute contraindication. As regards position and extent Zink found that even arrested lesions which extended beyond the upper lobe generally got worse. Carpi remarks that it is the position of the lesions rather than their grade which is of import; he says good results are obtainable only where disease is confined to the apex or the hilum of the lung; if at the base, especially at the left base in the region of the heart, they always tend to get worse. Forlanini, however, has described two cases with double lesions too advanced to treat, but which later developed spontaneous pneumothorax; this was kept up artificially, and resulted in great improvement in both cases, in one proceeding even to complete recovery. On this account he suggests that some cases with double lesions should be given the chance, and he and certain others have treated both lungs alternately with some measure of success.

Certain operators, also, have treated cases of double

disease simultaneously with at least temporary improvement in the general health and comfort. Fagioli succeeded in introducing gas into both pleural cavities in five among twelve cases of considerable double disease in which this was attempted; 100 c.c. to 150 c.c. of nitrogen was put in at longish intervals, the pressure being, of necessity, kept below the atmospheric. Under such measures he obtained notable improvement in three cases: temperature and pulse fell, the sputum diminished, the weight rose, and hæmoglobin increased greatly. In spite of this practical success Morelli condemns this method on the theoretical ground that in a double pneumothorax it must be the elastic healthy lung tissue which will tend to collapse, leaving the diseased and less elastic parts unaffected. Parry Morgan, however, points out that elastic tension along a lung axis containing diseased tissue must necessarily be increased; hence gas introduced into the pleural cavity will tend to collect over diseased areas, and by releasing this overstretching, particularly in inspiration, will allow the diseased parts to rest. This seems to the writer a likely occurrence where a minimal quantity of gas is admitted, as in Fagioli's cases, while Morelli's objection might hold if larger fillings were attempted. In any case it must be remarked that bilateral pneumothorax has not yet emerged from the experimental stage, and that for ordinary pneumothorax treatment the marked involvement of both lungs is for the most part a clear contraindication. Cases may improve for a time; Pisani recorded improvement in 33 of his 72 bilateral cases, and great improvement in the remaining 39, but in all it was but temporary. On the other hand, in some cases disease in the functioning lung may be stirred to increased activity and the inevitable end actually hastened.

The treatment of cases but doubtfully suited for artificial pneumothorax can but tend to discredit a method possessing quite definite indications within whose limits brilliant and lasting results can be con-

fidently anticipated. This is not to deny that these indications may be legitimately extended with increased experience—indeed, the writer is tempted to anticipate a time when temporary pneumothorax may become a recognised method of treating the vicious circle of auto-inoculation in cases where fever cannot be reduced by “typhoid” rest. But that time is hardly yet, and the credit of the method is best served by conforming at present to its well-recognised limitations.

Early Phthisis.—A disqualification, even more important in the writer’s opinion than that afforded by bilateral disease, exists in the instance of early phthisis. The application of pneumothorax treatment to these cases can be shown on many grounds to be contraindicated. In the first place it is unnecessary, since early phthisis is a disease readily curable by other methods; in the second place it is inexpedient since it brings with it risks and the chance of serious complications out of all proportion to the benefits it can confer. Pleural shock and gas embolism are, perhaps, but infinitesimal risks where modern methods are properly applied, but they cannot be altogether eliminated—particularly the former in cases of early disease. For de Carle Woodcock and Clark treating cases of this class have found pleural shock a not uncommon accident, and they especially note its greater frequency in early cases with a clear pleura than in patients with more advanced disease. This fact is in itself an important argument against the treatment of early pulmonary tubercle by artificial pneumothorax. Furthermore pleural effusion and the deformities which may follow it are complications which cannot in any way be avoided. Such effusions occur altogether in nearly 50% of cases, and though the shorter treatment required for slight disease would reduce this percentage, yet effusions must occur in many, and lung expansion fail or be incomplete in a few. Moreover, on re-expansion the pleural layers may become adherent, and the patient be thereby deprived of this treatment at a later stage when it may be truly indicated.

In some cases perhaps the production of a pneumothorax at an unusually early stage may be justified on economic grounds where the cure must be shortened in the case of a patient who is obliged to return to his occupation.

Having condemned, then, the extremes wherein pneumothorax treatment is resorted to too early or abandoned till too late, we may return to the recommendation of Forlanini to think of pneumothorax treatment as soon as ulcerative processes appear in the lungs. Cavitation of anything but small extent, an infiltrative process progressing in spite of a fair trial of sanatorium treatment, these, where the other lung will pass muster, are in general the indications in cases of ordinary phthisis. The acuter forms of tubercle need separate consideration.

ACUTER FORMS OF TUBERCULOSIS

Miliary tuberculosis does not, of course, come into consideration. The acuter broncho-pneumonic forms, whether for the most part apical or central, are commonly so far bilateral that the question of pneumothorax therapy hardly ever arises. Caseous pneumonic cases, on the other hand, are generally one-sided at first and have been submitted to pneumothorax treatment with varying success at the hands of different operators. Forlanini has not been fortunate in cases of this type, having found that disease always invaded the opposite lung. Brauer, and also Saugman, however, consider this type of disease suitable for pneumothorax treatment. The latter has had success in a case of consolidation of the whole of one lung; Dumarest and Keller also each claim a cure in a case of this class. Weiss treated five pneumonic cases; in four disease appeared on the opposite side, in one case as late as six months after, and death resulted. In the fifth case, which was one of caseous pneumonia, on the top of chronic phthisis, recovery occurred.

The writer has treated one such case, published in

the *Lancet* of July 15, 1916, with the best possible results up to the present. After eighteen months' pneumothorax treatment the patient is at work, in good general health with gain of over a stone in weight, and with the functioning lung free of disease. The pneumothorax is, of course, maintained. This is a satisfactory outcome if time confirms it; the writer has occasionally seen these pneumonic cases settle down to a quiet chronic process without pneumothorax treatment. Apart from the danger of active disease arising in the better lung in these cases, the pneumonic lung is, for mechanical reasons, but ill adapted to compression and collapse, or to processes of repair. This was particularly insisted on by Graetz, two of whose three autopsies exhibited pneumonic changes. These areas he found to be much less completely collapsed, and much less readily encapsuled and obliterated by the growth of fibrous tissue than are scattered, isolated tubercles.

HÆMOPTYSIS

Collapse of the lung may be indicated in cases of severe recurrent hæmoptysis, or as an emergency operation for a bleeding otherwise uncontrollable, and likely to prove fatal. Under the latter conditions a large quantity of gas, 1,000 or 1,500 c.c., may be put in at once, the needs of the moment outweighing the usual contraindications. Such large quantities are not, however, necessarily required to arrest the hæmorrhage. Masenti, Borgogno, and Vergano found that no great amount of collapse was needed, nor even a positive intrapleural pressure, to achieve this object. A gas quantity varying between 250 c.c. and 1,100 c.c. sufficed to stop the bleeding in their five cases. If a small amount is found sufficient, further collapse may be deferred to a second filling, a complete pneumothorax being indicated where the other lung is in a condition to stand the strain. In these cases, however, it will often happen that the other lung is markedly diseased,

and only partial measures can be undertaken. Pleural adhesions, moreover, may restrict or altogether prevent the operation; in a case of the writer's treated for recurrent hæmorrhage, only a partial pneumothorax could be attained, and this reduced but did not arrest the recurrent attacks. To effect a permanent cure in such cases the responsible cavity must be completely flattened out and maintained so under a suitable positive gas pressure.

SPECIAL CONTRAINDICATIONS

Disease of various organs, whether tuberculous or not, may contraindicate the production of an artificial pneumothorax.

Lungs.—The condition of the functioning lung, *qua* tuberculosis, has already received consideration. In addition advanced emphysema and asthma are contraindications. Dyspnœa may be toxic, and in this case will be relieved by the removal of autoinoculation; if dependent on widespread lung involvement or emphysema, when some degree of cyanosis will be detectable, it is, of course, a clear contraindication.

Circulatory Organs and Kidneys.—Pneumothorax therapy probably reduces the pulmonary vascular area, in most cases at least, and thus puts a strain on the right heart. In addition the loss or reduction of intrathoracic suction must tend to embarrass the venous circulation. The effect of these disabilities on circulatory organs and kidneys renders any serious disease of these organs a contraindication to pneumothorax treatment. On the other hand it must be noted that certain cases of nephritis appear to be of tuberculo-toxic origin, and have been known to recover when autoinoculation was removed.

Laryngeal Tuberculosis is no contraindication unless far advanced, and even then a doubtful one. Da Gradi has described three bad cases from Forlanini's clinic which improved and eventually healed under pneu-

thorax treatment. Slighter cases of laryngeal disease commonly improve; eleven such cases mentioned by Zink all recovered except one very severe one, which nevertheless decidedly improved.

Intestinal Tuberculosis is a contraindication unless very slight and consequently latent. Carpi records that six among his thirteen fatal cases died of intestinal tuberculosis, which became more active under pneumothorax treatment. Brauer and Spengler, however, record two cases where intestinal disease caused symptoms, and where these entirely disappeared under pneumothorax treatment. Forlanini is not against the inclusion of slighter cases.

Diabetes.—The presence of this disease should make the physician very wary of recommending pneumothorax treatment; not only are latent foci in the better lung very likely to become active and progress, but the punctures may inflame and give trouble as in a case recorded by Weiss.

CHAPTER III

PNEUMOTHORAX APPARATUS

GENERAL FEATURES

THE essentials of a pneumothorax apparatus are surprisingly small and simple—no more, in truth, than could be collected for emergency in many a doctor's surgery, and put together in half an hour or less. A couple of wide-mouthed bottles of about 1 to 1½ litre capacity, a few feet of glass tubing of about 4 mm. internal diameter, and some rubber tubing of approximately equal bore—these constitute the main requirements. The corks of the bottles, which must be of rubber if for more than temporary use, must be bored in two places; the gas bottle should be ruled so as to record volumes in 100 c.c. or less, and numbered from below upwards.

The bottles are half filled with water, or better, an antiseptic solution (p. 37), and are put into connection at their lowest point by tubing reaching to the bottom of each, whereby the fluid can be made to flow from one to the other by an appropriate alteration of their levels. The air above the fluid is in one, the "pressure bottle," open to the outside air through a short piece of glass tubing, in the other, the "gas bottle," it is connected by a glass T piece with the manometer on the one hand, and with the pneumothorax needle on the other (fig. 1). A cotton wool filter of some kind must be introduced between the T piece and the needle to filter the air or gas. This can be a small glass chamber, corked at each end, or a length of stout glass tubing blown into a

bulb, and packed with sterile wool. The manometer consists of two uprights of glass tubing some 40 cm. long connected together below by a short length of rubber tubing. Water is entered to a point about half way up each length, and a scale ruled in half centimetres fixed between them and numbered 1, 2, 3, 4, etc., upwards and downwards from the level of the water. Readings are made only on the limb which is open to the outside air, and, since each half centimetre rise (or fall) is accompanied by a half centimetre fall (or rise) of the opposite column of fluid, the readings on the scale will record the difference between their levels, that is the pressure, in centimetres of water.

These are the essentials of a pneumothorax apparatus, and the nearer the instrument can be kept to these primitive lines the better it is likely to answer its purpose. Every departure from them must, for its justification, be shown to satisfy some important need. The apparatus designed by Vere Pearson and Lillingston and shown on p. 26 is here recommended on the ground that it departs so little from first essentials, and yet satisfies all practical requirements. The gas bottle has been made long and narrow, so that the slightest change of fluid level can be quickly noted, and small quantities of gas measured with fair accuracy. The manometer is conveniently fixed to a firm partition, and its upper ends are bulbous, so as to minimise the risk that the fluid be unexpectedly sucked or forced out of the ends. A small screw clamp surrounds the connecting rubber at the base, and thereby the respiratory fluctuations can be abolished at will, and a mean intrapleural pressure recorded. The tall graduated bottle is filled with nitrogen from a cylinder, or with air, the fluid, generally an antiseptic solution, being driven back into the pressure bottle, and retained there by clamps on the tubing. When the clamps are released the fluid will flow from the higher level by syphon action, displace the nitrogen, and force it at any desired pressure through the pneumothorax needle,

This apparatus is a good example of movable flask apparatus in general, and is similar in design to those used by Murphy, by Brauer, and by Spengler. These

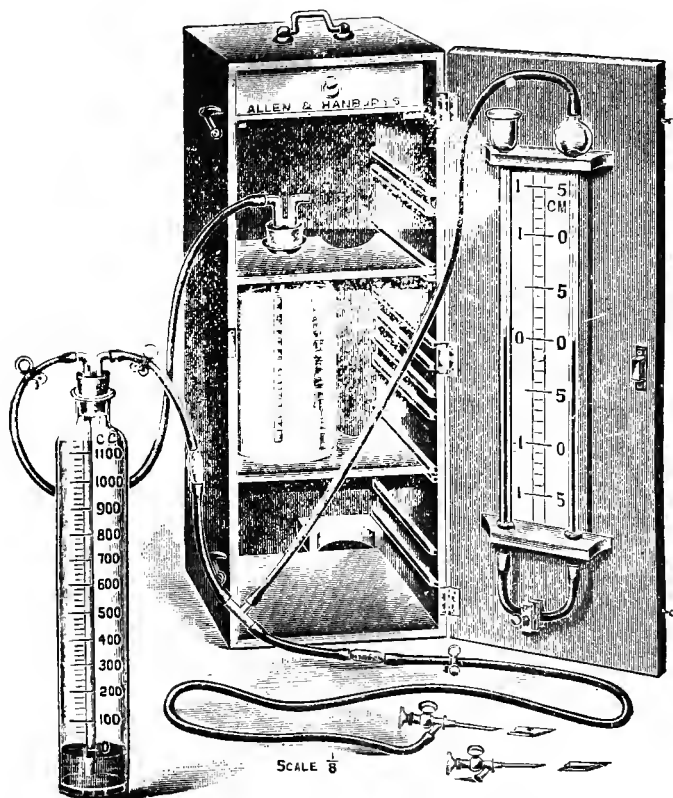


FIG. 1.—PEARSON AND LILLINGSTON PNEUMOTHORAX APPARATUS.

The manometer is better graduated in half-centimetres (for reasons given on p. 25) and numbered 1, 2, 3, 4, 5, etc., all the way up and down for facility of reading.

operators, however, appear to have employed flasks of 2-litre capacity, which would seem needlessly cumbersome and heavy. Convenient but more elaborate and expensive apparatus on somewhat similar lines are those

of de Carle Woodcock and Lister in this country, and of Küss of Paris. Woodcock's manometer consists of a narrow tube immersed in a larger reservoir of fluid, the upper end being continued into a T piece connected on one side with the needle, and on the other with the gas bottle. The bottles move up and down on upright metal rods, the nitrogen is manufactured by sucking the air through a pyrogallate solution, and in Lister's modification of this instrument an oxygen apparatus is also added. Lister has also designed a regulator with a dial whereby the different parts of the apparatus can be mechanically connected or cut off with ease and certainty. Küss's gas bottle is fixed at a somewhat elevated level, while the pressure bottle is made to move up and down on a rack and pinion; the nitrogen is kept in a bag from which the gas bottle can be supplied whenever its contents have been replaced by fluid.

A word must be said about apparatus with fixed bottles, since this is still used by such authorities as Forlanini, Saugman, and v. Muralt. The pressure and gas bottles and the manometer are all fixed on a stand, and when prepared for use the fluid is driven back into the pressure bottle by connection with the nitrogen cylinder. When the needle is in the pleural cavity the fluid pressure is released, and forces the gas as slowly as desired into the chest. But this fluid pressure, since the apparatus is fixed, must be high at first, and must steadily fall as the fluid descends into the bottom of the gas bottle. Soon a point is reached when the intrapleural pressure balances the fluid pressure, and the flow ceases altogether. The pressure must then be raised by the use of a hand bellows, an ill-regulated and necessarily intermittent source of power, which Morriston Davies in this country has advantageously replaced by the use of an accessory pressure bottle. The disadvantages of fixed bottles are manifest, and are especially apparent at a first operation, where it is advisable, in the interests of safety, to enter gas at first at a decided negative pressure. This is, of course, easily accom-

plished with movable bottles by raising the gas bottle so that the level of its fluid is the desired amount above that in the pressure bottle. Saugman with fixed bottles tries to overcome the difficulty by starting with the fluids at equal level, but is soon driven to use the hand bellows to secure the admission of gas. A position equally disadvantageous is experienced when a Replacement of Gas is indicated in cases of pleural effusion (p. 147). With fixed apparatus the admission of gas into the pleural cavity in this operation must almost necessarily be intermittent, since undivided attention must be given to its entrance under an uncontrolled fluid pressure, whereby undesirable or even dangerous intrapleural pressures may inadvertently be reached. With movable bottles, on the other hand, the gas can be allowed to flow all the time the fluid is draining away, all that is required being an occasional raising of the pressure bottle to maintain the slight positive pressure generally desired. For these reasons, among others, the writer does not hesitate to raise his voice most strongly in favour of one or other form of movable bottle apparatus, and against all apparatus with fixed cylinders.

Having dismissed the gas and pressure bottles or cylinders about which, beyond their mobility, there is not much to be said, a little more attention may be devoted to other parts of the apparatus.

THE MANOMETER

The water manometer should be some 35 to 40 cm. high, and made with bulbous upper extremities, as already noted, so that the fluid will not be so readily driven out by cough, nor drawn into the chest under an exceptionally great negative intrapleural pressure. The calibre of the manometer tubing is, in the writer's opinion, of some importance for the recording of those delicate fluctuations of pressure which keep us in touch with intrapleural changes. The wider is the discrepancy between calibre of needle and size of manometer tubing,

the less responsive is the column of fluid. Thus the respiratory fluctuations on various occasions in the case of *H. B.* with a fine Brauns' needle (calibre .8 mm.) showed—

Before Filling.		After Filling.
2 cm. H_2O .		2 cm. H_2O .
2 " "		1 " "
4 " "		2 " "

but with a Saugman needle (calibre 1.2 mm.)—

Before Filling.		After Filling.
7 cm. H_2O .		4 cm. H_2O .
6 " "		3 " "
5 " "		3 " "
6 " "		2 " "

a manometer of 4 mm. internal bore being used in all cases.

Where the Saugman needle was retained but a manometer of large (10 mm.) bore substituted, the fluctuation was again diminished thus:

Before Filling.		After Filling.
4 cm. H_2O .		2 cm. H_2O .

The comparative inertia of the fluid in a manometer of so large bore is very obvious in practice, and the clean and ample fluctuation which is such reassuring evidence that the end of the needle is in the pleural cavity hardly shows itself with a needle of ordinary calibre. The fluctuation is somewhat reminiscent of that obtained with a half-blocked needle. Saugman uses manometer tubing of 3 mm. bore. The writer has found 4 mm. internal diameter gives very satisfactory results, and would advise against the use of any exceeding 5 mm. It must be borne in mind also that the chances of gas embolism are increased by the use of tubing of wide bore, since the amount of gas released at the needle point at the moment of a negative fluctuation must thus be notably increased (p. 159). To reduce still further the risk of gas embolism Würtzen introduces at first operations a small water manometer close to the

needle, so that rarefaction of gas in a length of tubing is avoided and a fluctuation of — 14 cm. only provides 1 c.c. of gas at the needle point.

It has become customary in pneumothorax work to measure intrapleural pressure in centimetres of water, since the water manometer is that almost invariably used in practice. Equivalents in mm. of mercury may be marked on the scale if desired, and some apparatus, as those used by Brauer, by Saugman, and by Hansen, possess a mercurial manometer in addition for the recording of very high or low pressures. Obviously 35 to 40 cm. is the limit of height for use in a convenient and portable apparatus, and positive pressures above that point will rarely, if ever, be required. It may happen, however, that a very large negative pressure may have to be expected in cases where fillings have been neglected or purposely discontinued, and in this event the use of a mercurial manometer will save the risk of the manometer fluid being sucked into the chest. Forlanini overcomes this difficulty by the provision of an air chamber, in connection with the open end of the manometer, of such known capacity that the pressures can be thereby doubled. During ordinary use it is kept open to the air, but when its mouth is closed by a tap, the manometer readings are suitably controlled by the presence of this column of rarefied or compressed air.

It is an essential of proper treatment or even of safety that the manometer be never shut off from the needle, so long as this is in the pleural cavity. That this warning is not needless is illustrated by the remark of a recent writer that the pressure after a filling in one of his cases must have been high, since on opening communication with the manometer the manometer fluid was blown out of the tubing. Such an occurrence can only be condemned as malpractice.

THE GAS TO USE

The question of a suitable gas revolves about the properties of Oxygen, Nitrogen, and Air; the suitability of fluids such as normal saline or oils for injection can be dismissed as, at the present time, irrelevant. Choice of a gas is open to discussion on two main grounds—the question of safety, that is, the risk of gas embolism, and the question of diffusibility.

At a First Operation.—A good many operators, notably Saugman, employ oxygen at the first filling with a view of avoiding the risk of gas embolism. Their argument is that oxygen is more readily absorbed by blood than is nitrogen, and if it is venous blood that is in question, this reasoning cannot be gainsaid. But it must be borne in mind that serious embolism can but rarely occur through the wounding of veins containing venous blood, namely those in the thoracic wall (see p. 156). It is from the lung that the danger of embolism is real, and the pulmonary veins contain arterial blood, which has, as Brauer reminds us, no special power of absorbing oxygen (p. 159). There is then, in reality, no decided advantage of Oxygen over Air or Nitrogen at a first operation, more especially since, if a pulmonary vein is wounded, embolism must be at least as easily produced by alveolar air at atmospheric or at times a slight positive pressure, as from the apparatus gas at a distinctly negative pressure (see p. 158). The writer has always given air or nitrogen at first operations, and relies on other precautions for the avoidance of gas embolism.

At Refills.—For refills oxygen gas is unsuitable on account of its high diffusibility—the choice must lie between the highly inert gas nitrogen and air in which nitrogen is diluted with 21 vols. per cent. of oxygen. Nitrogen has come into extensive or almost universal use, but it is doubtful whether its slight advantage over air entirely justifies the trouble and expense of obtaining

it. It has been made clear by experiment that whatever gas is put into the pleural cavity its composition is in a short time entirely altered by exchange with the gases in the blood and by the effect of the local respiration of the tissues. This was first recognised by John Davy as long ago as 1823. Having found that the gas in a case of spontaneous pneumothorax consisted of a mixture of N_2 92% and CO_2 8%, and in another case of N_2 93% and CO_2 7% he enquires—"What had become of the oxygen that had disappeared; whence the carbonic acid gas with which the azote was mixed?" To resolve these difficulties he conducted on dogs numerous observations designed to test the absorption of various gases from the pleural cavity. Of recent years William Ramsay, analysing the gas in a case of pyo-pneumothorax treated with oxygen replacement by Morrision Davies, found the pleural content after the withdrawal of 1,250 c.c. of fluid and introduction of 750 c.c. of O_2 to be— N_2 74.09%, O_2 21.56%, CO_2 4.35%. Four days later the oxygen had been practically absorbed and replaced, the content being— N_2 98.13%, O_2 0.69%, CO_2 1.18%. Tobiesen of Copenhagen gave pneumothorax fillings of air, oxygen, nitrogen, and carbon dioxide, and by the next refill he always found the same mixture present, namely N_2 90%, CO_2 6-7%, and O_2 3-4%. Webb, Gilbert, James, and Havens found that within 24-48 hours in cases filled with nitrogen, O_2 4% and CO_2 8% had appeared on the scene, and with air a precisely similar result occurred, showing that some 15% of oxygen had diffused into the blood at the same time that the CO_2 had come from the blood. When oxygen is used for a filling, this remains in larger quantity, as O_2 14%, CO_2 6% and N_2 80%, though obviously replaced to an enormous extent by other gases. The authors argue that owing to this rapid diffusibility of gases, there can be no advantage of nitrogen over air in treatment by artificial pneumothorax. Though these various observations go some way to support this conclusion, they cannot show to what extent in the

respective cases the lost gases had been replaced, and hence how far the total volume was diminished after the 24-48 hours' interval. Notably it could not be said how much, if any, of the lost 15% of O, when air was used, was replaced by fresh nitrogen, nor whether the total loss was not in this case, on account of the high diffusibility of oxygen, decidedly greater than in the case where nitrogen alone is utilised. The question is whether, in practice, the presence of some 21% of a more diffusible gas with the nitrogen shortens the efficiency of a refill to any recognisable extent. This question may be answered to some extent at least by the following record of refills from a case under the writer's care:—

Mr. P. W. P. :

3.1.15	. - 10 - 1	620 c.c. N.	- 4 + 5
22.1.15	. - 10 to 0	660 c.c. N	- 5 + 5
11.2.16	. - 10 - 1	600 c.c. N	- 4 + 5
3.3.16	. - 9 - 1	560 c.c. N	- 4 + 5
24.3.16	. - 8 to 0	460 c.c. Air	- 4 + 5
14.4.16	. - 7 + 1	400 c.c. N	- 4 + 5
5.5.16	. - 8 + 1	490 c.c. N	- 3 + 6
26.5.16	. - 8 to 0	520 c.c. Air	- 3 + 5
16.6.16	. - 8 - 1	560 c.c. N	- 3 + 5

Here we see, and the same can be shown for other cases, that the efficiency of air and nitrogen, as measured by their absorption in identical intervals of time, was hardly, if at all, distinguishable. Indeed for practical purposes air can be made to replace nitrogen for refills whenever convenience may dictate, and the writer has almost abandoned the use of the latter gas during the past nine months. This does not, however, apply to complete gas replacement of fluid (see p. 143), where the presence of this amount of oxygen must exert a decided influence on absorption.

TUBING

The apparatus, including the tubing, must be sterilised before putting it together for use; afterwards the

inside of the tubing below the filter, the only vital part, can be kept sterile most conveniently by closing the end with a glass rod as in Pearson and Lillingston's apparatus. An addition which should never be omitted is a small length of glass tubing inserted close to the needle; this forms a window through which fluid may be seen should any enter the tubing in a case of pleural exudate (p. 150), and also blood under exceptional circumstances in the case of a wounded vessel.

NEEDLES

A large variety of needles have been used by different operators for the introduction of gas into the chest. For practical purposes they may be divided into those suitable for a first operation and those suitable for refills, since the indications in these two cases are somewhat different.

For Initial Operation.—The requirements of a needle for the first production of a pneumothorax are somewhat complicated. It must be capable of piercing the skin and superficial structures with ease, but it should be blunt at the moment of its passing the parietal pleura

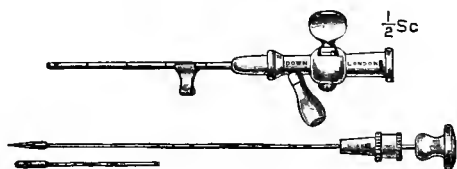


FIG. 2.—WRITER'S TROCAR AND CANNULA FOR FIRST OPERATIONS.

so as to avoid any risk of wounding the lung. These requirements are best satisfied, in the writer's opinion, by the use of a trocar and cannula, such as that depicted in fig. 2. This instrument is provided with an obturator to prevent the admission of air; the cannula of 1·8 mm. (·072 inch) gauge has a side opening near the tip, is graduated in centimetres, and carries a sliding guide to

regulate the distance it is to be thrust into the chest wall. With the skin held stretched the sharp trocar and cannula are pushed in up to the guide fixed at 1 cm. or less from the point (see p. 44), or till the shoulder of the cannula is well through the skin. The trocar is now drawn out into the obturator, the tap of the needle closed and obturator and trocar removed. The unarmed cannula, whose edge should, however, be sharp, is now carried forward through the parietal pleura. Küss pushes a cannula and sharp trocar down to the external intercostal muscle, and then replaces the latter by a blunt stilette for its further passage into the pleural cavity. The blunt stilette is grooved on one side so as to bring the side opening of the cannula into connection with the manometer. Murphy used a trocar and cannula, but in addition notched the skin with a tenotome; Morriston Davies in this country adopts a similar procedure for the introduction of an obtuse-ended hollow needle. Other forms of blunt instrument in occasional use are those of Courmont, Adolph Schmidt, and Deneke.

Many operators make use, both at initial operations and refills, of fine hollow needles with a point cut obliquely so as to form a cutting edge, whereby the danger of wounding the lung is notably reduced. Of these the needles of Forlanini and Brauns and Sahli are examples. The former uses for ordinary purposes a needle of 1·1 to 1·2 mm. external diameter, but in cases under a high positive pressure needles as small as ·8 mm. to ·4 mm. He claims, on experimental grounds, that injury to the lung never occurs with the use of these needles in his own hands. The writer has made extensive trial of a Brauns needle of ·8 mm. (·032 inch) external diameter, but has found it so frequently become blocked, either with a particle of punched-out epidermis or with a drop of moisture, that he has finally abandoned the use of fine-calibred needles. Three centimetres is a sufficient length for an instrument used for first operations, but for refills a length of 5 centimetres will be needed on occasion.

For Refills.—When once a gas collection has been formed, there is no longer danger of wounding the lung. A sharp pointed hollow needle is then indicated, whereby the chest wall can be pierced with ease. The needle should not be too small in bore (see above), and the Saugman needle of at least 1·2 mm. (.048 inch) external diameter answers the purpose sufficiently well. This may be provided with an obturator, as is done in the most modern pattern, or the end may be covered, to prevent the entrance of air while clearing, by a thin rubber cap, through which the stilette is passed. There is, however, the disadvantage with this needle that it may punch out a piece of epidermis in its passage through the skin, and carry this with it into the pleural cavity. This can be avoided by the use of a solid

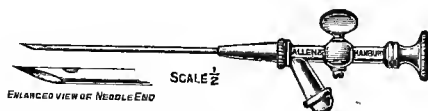


FIG. 3.—THE LILLINGSTON NEEDLE.

Identical with the Saugman needle, but with the terminal opening closed with metal.

pointed needle with a side opening, such as that of Kjer-Petersen used by Saugman himself at refills, or in this country the Lillingston needle, which is practically a Saugman with the end opening filled in and a side opening provided in its place (fig. 3). These needles, if properly dried before operation and if the local anæsthetic is allowed a few minutes for absorption, practically never get blocked at refills in a dry pleural cavity. Consequently the obturator and stilette are but seldom needed, and with the older pattern of needle the writer dispenses with the rubber cap on all but rare occasions. The stilette must, however, never be passed without protection from the outer air being provided by one or other of these means.

The method of open incision down to the parietal pleura introduced and practised by Brauer can be dis-

missed without further description, as it has now been almost universally abandoned.

The "safety syringe" of Forlanini needs a word of reference; it is made to fit on to the posterior free end of the needle and, by the use of suction, to exclude at a first operation the chance that the end of the needle has entered a vein. Saugman has rightly condemned it as liable to lead to blocking of the needle and, indeed, if the characteristic manometer fluctuation is always waited for as evidence of the position of the needle point, the safety syringe should at best be a superfluity. The only condition under which its use may be recommended is that where a large pleural effusion is to be replaced with gas, and the manometer fails to register, owing to the needle becoming blocked with fluid. Under these circumstances the suction of some fluid into the syringe supplies the required proof that the end of the needle is in the pleural cavity (see p. 150).

PREPARATION OF THE APPARATUS FOR USE

To prepare the apparatus for use, the tubing is sterilised in boiling water, and afterwards carefully drained and dried; the bottles are filled with an antiseptic solution, such as 1% or 2% Izal or Lysol, or 1 in 1,000 perchloride or biniodide of mercury. When this has been accomplished and the apparatus again put together, the bottles should be firmly corked with the gas bottle still full of fluid, but the other emptied of all but enough to cover the lower end of the longer glass tube, a clip being placed on the rubber tubing between them. To fill with gas, the bottles should be detached from the manometer, and the fluid in the gas bottle replaced by nitrogen in the following manner. The taps of the nitrogen cylinder are first turned on so that gas escapes in a slow stream; the clip is taken off the tubing between the bottles and the rubber tubing leading to the upper end of the gas bottle is then connected with the cylinder. The fluid is thus driven back into the pressure bottle

and replaced by gas. At the end of this operation the gas must be brought to atmospheric pressure, either by opening the tubing to the air, or by bringing the fluids to the same level before fixing on the clip between the bottles. If air is to be used, it is sucked into the gas bottle by syphon action through a suitable alteration of the fluid levels. It has been recommended that the gas bottle should be filled twenty-four hours before use, to allow dust particles to settle: most conveniently it is refilled at the end of an operation so as to be in readiness for the next. The tubing for the gas bottle should be re-connected with the manometer tubing before it is put away so that their inner parts may be protected from dust contamination.

The interior of the tubing connecting the cotton wool filter with the needle is kept sterile, as already mentioned, by the closing of its extremity with a glass rod. It and the filter should, however, be resterilised from time to time, and in particular after use in a case where fluid from the pleural cavity may have entered its end. It is well to have a spare tube ready for use in cases with pleural effusion.

The needles, both pneumothorax and hypodermic, must be always kept clean and sharp, and a small hone, specially kept for this purpose, is an indispensable requirement.

CHAPTER IV

TECHNIQUE OF INITIAL OPERATION

NURSING HOME OR PRIVATE HOUSE

THE operation for artificial pneumothorax can be quite easily performed in an ordinary bed and bedroom in the patient's own home. Nevertheless there is one important advantage which a Nursing Home or Hospital may have over a private house, and that is the presence of an X-ray installation. If this advantage can be obtained it is an important gain, for the X-rays during the early weeks of treatment are of very great assistance in supplementing the information derived from physical signs, particularly in deciding on the presence and position of adhesions, and in determining the moment when satisfactory collapse of the lung has been obtained. Nevertheless treatment can be carried through at these stages, and even altogether, without any use of the X-rays, and the writer has had to submit to this experience on many occasions. Forlanini has remarked: "The clinical examination alone suffices, in the absence of radioscopic apparatus, for the administration of the treatment." If treatment is carried out in the patient's home, the presence of a trained nurse is an almost indispensable convenience during the first few weeks—later she can be dispensed with, and finally the patient will be able, in most cases, to attend for refills in the doctor's consulting room.

PRELIMINARIES

The patient should be kept in bed for some days prior to the initial introduction of gas—this will probably be

indicated on other grounds, since the pneumothorax case is generally a febrile case, "ambulant febrile" at least. During this time the temperature, pulse, and daily amount of sputum should be recorded on a four-hourly chart. It is convenient, also, during these preliminary days, to examine the chest carefully with a view to the selection of likely sites for puncture. These should be duly noted in order of their suitability so that no needless delay may occur on this account at the time of the operation.

SELECTION OF SITES FOR PUNCTURE

The possibility of producing an artificial pneumothorax depends on the discovery of free pleural space, and the comfort of the patient demands that this object should be attained with as few punctures as possible, and preferably at the first. In the selection of sites for puncture, certain facts must be borne in mind. Firstly, the base is a much more hopeful region than the apex, both because it is farthest from the disease area, and because the free movement of the lung border tends to keep its surface free. Moreover the axilla is a more likely spot than the front or back of the chest, largely because the respiratory excursion is the greatest at this point. This respiratory excursion, the tidal movement of the lung border between deep inspiration and expiration, or deep inspiration and quiet breathing, should be marked and measured in front, behind, and in the axilla with a view to deciding on which surface pleural adhesions are least likely to be present. In this connection it must be remembered that the lung excursion differs normally at these three points, being largest in the axilla (10 to 12 cm. difference between forced inspiration and expiration), smallest behind (3 or 4 cm.), the front being of somewhat wider extent than this latter. If a free tidal movement is present, the production of a pneumothorax is practically assured—if, as often happens, there is little or no tidal fluctuation, the discovery of

free pleural space at some point is still possible since the limitation of movement may depend on adhesions above the base, or on diminished expansile power of the lung itself.

The axilla, often the spot most free of pleural adhesions, has also the advantage that the intercostal spaces are wide and that the chest wall between the pectoralis and latissimus dorsi is but thinly clothed with muscle, and hence presents an easy and convenient site for puncture. The writer is accustomed to make a first puncture in this region, generally at the edge of the lung, as for instance in the sixth space in the anterior axillary line, unless there exist special reasons against it. Thus in some cases with marked basal disease it may be fairly certain that adhesions are present in this neighbourhood, and a better chance may offer itself at a point higher up the chest wall, often in the second or third spaces in or outside the mammary line. The point farthest from the older foci of disease and where signs of disease are absent or least marked is the point where free pleura is most likely to be found. Such points are generally near the base, and if a couple of punctures in the subaxillary region, one in front of this, and one at the back, all near the lung border, fail to find a pleural space, it is very unlikely that further attempts will be successful. Saugman remarks that a useful pneumothorax is generally produced at the first puncture, rarely at the second to fourth, and never after this.

Deneke has reminded us that the presence of pleural friction is evidence of the absence of adhesions. In a couple of cases with widespread adhesions the writer has been helped to success by the presence of a small amount of pleural fluid at the base. In such cases it will generally be found that change of position exerts a certain slight influence on the position of the fluid, and if the needle can be entered at a point where the dulness of fluid has been, by change of position, replaced by resonance, a successful puncture is practically assured. The neighbourhood of cavities, particularly

superficial cavities, is quite unsuited for puncture, partly because strong pleural adhesions are almost sure to be present, and partly because there is risk that, if the puncture fails, the needle may wound the cavity wall, and this wound be opened up into the pleural space if the operation succeeds elsewhere, and the adhesions at this point are subsequently broken down. The same danger applies to caseous areas and areas of marked consolidation, the wounding of which is likely to favour a perforation of the lung, if an artificial pneumothorax is finally achieved.

THE OPERATION

Preparation of Patient.—Do not operate after a large meal. The procedure must also be so arranged as to avoid the period of the catamenia in women, since nervous hypersensitiveness at these times heightens the risk of pleural shock. A nervous patient may be treated with bromides as recommended by Kuthy, and some diffusible stimulant as alcohol or Spiritus Ammoniae Aromaticus may be given just before the operation. About half an hour to one hour before the initial operation, an injection of Morphine (gr. $\frac{1}{8}$ – $\frac{1}{4}$) with or without Atropine (gr. $\frac{1}{100}$), or Omnopon (gr. $\frac{1}{8}$ – $\frac{1}{3}$), a satisfactory preparation for these cases, must be given.

Preparation of Instruments and Apparatus.—A low table covered with a clean towel must be placed beside the head of the patient's bed. The pneumothorax apparatus is stood thereon wide open and with the manometer turned to face the operator; the bottles are taken out and, if not previously filled (as is best so as to allow dust to settle), are now charged with N gas or air. The tubing is seen to be properly joined up, and the long tube to which the needle will be attached is coiled into a bowl of 1 in 20 carbolic, its end being closed by a glass rod. In addition the following instruments must be at hand :

Tincture of Iodine and some wool swabs and dressing forceps to apply it.

Sterile towels.

A sterilised hypodermic syringe with two fine and sharp sterilised needles.

A bottle or ampoule of sterilised 2% Novocaine in normal saline, or in 1 in 10,000 Adrenalin solution.

The Pneumothorax needle or needles lying in absolute alcohol.

A bowl of hand lotion.

Ether and other emergency remedies (see p. 155), and a spare hypodermic syringe and needle to apply them if required.

Position of Patient.—The patient lies supine, but turned somewhat on to the sound side, so as to bring the proposed site of operation directly uppermost (see also p. 65). Saugman makes a great point of having the area of operation highest, so that a gas bubble, with its tendency to float upwards, may collect round the needle point before this can reach the lung. But it seems clear that the gas from the manometer tubing remains round the end of the needle for other reasons, since otherwise the respiratory fluctuation characteristic of a successful pleural puncture would not occur in other apparently less suitable positions, and this is not the case.

The head is kept low to reduce the risks of gas embolism, and a small firm pillow is placed under the back and sound side, so as to stretch and open out the intercostal spaces round the seat of operation. The arm is stretched above the head and may, if required, be supported by a nurse. The selected site or sites of operation are now found and marked with a skin pencil, and at the same time it is expedient and convenient to mark out the border of the heart and the lower limit of the lung, so as to keep the anatomical boundaries in clear view.

Passing the Needle.—The operator now sterilises his hands and places a sterilised towel round the site of

operation, while an assistant or nurse swabs a wide area of the chest wall with Iodine lotion. He then fills the hypodermic syringe with novocaine, places it on the sterile towel, and takes the pneumothorax needle and stilette out of its alcohol. These he proceeds to dry thoroughly by heat in the flame of a spirit lamp, heating particularly the needle end, and passing the heated stilette several times through its lumen, so as thoroughly to dry it. It is essential that the needle contain no atom of moisture when it is ready for use. The needle is now placed on the sterile towel, the operation spot selected, and the novocaine syringe taken in hand. A small superficial, intracutaneous bleb is first made with the syringe sloped. The needle is now moved to the upright position without withdrawing it, passed into the intercostal space near the upper border of the rib, and pushed forward very slowly in fractions of a centimetre, some novocaine being forced out at the end of each move forward. Enough must be left in the syringe to anæsthetise the parietal pleura, the whole or main object of this procedure, and this will be found at a depth varying from 1 to $2\frac{1}{2}$ or even 3 cm., according to the site and the patient's nutrition and muscular development. In a thin patient the pleura may be only a bare centimetre from the skin. If the anæsthetisation is done sufficiently slowly, and it should take $1\frac{1}{2}$ minutes at least, no pleural pain may be felt—more often a sudden pain is experienced when the pleura is reached. Rarely, it appears, an intercostal nerve may be injured, and cause pain and tenderness of a more widespread and lasting character. The hypodermic needle is now withdrawn, and the pneumothorax trocar and cannula is fitted on to the end of the proper tubing, which is removed from the bowl of carbolic lotion for this purpose. The guide on the cannula if used is placed at about $\frac{3}{4}$ cm. from the point, the trocar kept firmly in place by pressure of the hollow of the hand on its upper end. With the first and second fingers of the left hand the skin of the anæsthetised point over the intercostal space is held tightly

stretched to each side, and the trocar and cannula are entered swiftly and firmly, but without any stabbing movement, up to the guide, noting carefully that the shoulder of the cannula is well through the skin before the trocar is withdrawn. The trocar is then pulled back into its stuffing box, the tap closed, and trocar and stuffing box removed. The cannula is now driven forward by careful, steady pressure, attention being closely fixed all the time on the manometer, and the final "snap" which announces perforation of the parietal pleura being carefully looked for. Before this is reached a painless resistance is often experienced at the inner muscular fascial layer; the patient generally experiences some pain at the moment when the pleura is reached and pierced.

Manometric Response.—Just outside the parietal pleura a feeble respiratory fluctuation about the atmospheric level may be observed. At the moment the end of the cannula enters the pleural space, the manometer fluid gives a sudden swing to a negative pressure of some 10 cm. of water, and continues round this point with clean respiratory fluctuations of some 4 to 8 cm. Such a clear indication of a high negative pressure with large respiratory fluctuations is complete proof that the end of the cannula is in the pleural cavity, and N gas can be entered forthwith.

Entrance of Gas.—This is done at a negative pressure about one-half to two-thirds that in the pleural space, a measure which is accomplished by so placing the bottles before the operation that the column of fluid in the gas bottle stands some 7 or 8 cm. higher than that in the pressure bottle. The gas is thus sucked into the pleural cavity at a slight negative pressure, as soon as the clips on the tubing are removed, a manœuvre which probably reduces the very small risk of gas embolism to a minimum. As soon as a definite rise in the level of fluid in the gas bottle has demonstrated the fact that gas has entered, the fear of embolism may be finally set aside, and the bottle fluids placed at the same level, so that the gas flows in under ordinary atmospheric pressure.

Amount of Gas at first Operation.—At a first operation it is convenient to put in enough gas to maintain an adequate supply against the next puncture and “refill.” Where the gas goes in easily 300 c.c. is a convenient quantity, and this should be put in in all cases where no pain or distress occurs, and where the intrapleural pressure is not brought above atmospheric pressure. Saugman recommends 200–250 c.c. and never above 300 c.c. at the first operation; nevertheless the writer sometimes gives 400 c.c. where the succeeding interval has to be as long as four days and where this amount of gas disturbs the pressure but little, and he has never experienced any ill effects from it. With a free pleural cavity these quantities of gas suffice to raise the pressure but little—it remains markedly negative (see Case of W. N., p. 57). Where only a moderately free pleural chamber is present the pressure may rise to the atmospheric (Case of H. B., p. 55). Where, on the other hand, only a small pleural pocket is opened up, the pressure may rise above the atmospheric after only 50 or 100 c.c. or little more has entered. With so small a cavity a rise of pressure even greatly above that of the atmosphere will cause no disturbance, unless a certain amount of pain, and in such cases the convenience of getting in at any rate 100 or 200 c.c. must be duly weighed against the patient’s discomfort. The ease and safety of the next puncture depend largely on the certainty of finding an adequate gas pocket into which the needle may be entered without fear of wounding the lung.

In the early days of pneumothorax therapy it was customary to enter a large volume of gas, 1,000 c.c. or more, at the first filling, but this has been generally abandoned as at least inadvisable. In cases even where a large quantity appears to be readily borne at the time, it has commonly happened that symptoms of distress, dyspnœa, sweating, and the like have followed after an interval, and a smart febrile reaction on the following day. Apart from this, also, there is a very

real danger of infecting the other lung by the large quantity of sputum squeezed out of the collapsing cavities and tubes of the diseased lung—a quantity too large to be immediately and safely removed by coughing. Zink, who formerly employed 1,000–1,200 c.c. at first fillings, records no less than 280 c.c. of sputum coughed up by one of his patients. Robinson and Floyd have recommended a first filling of 600–1,000 c.c. with the object of encouraging the patient by the rapid improvement of symptoms which is likely to follow it. In cases where the pneumothorax operation is undertaken for hæmoptysis, an immediate partial collapse of the lung is aimed at, and a large quantity of gas, 1,000 c.c. or more, must needs be run in without delay.

Manometer Readings.—During the time that the gas is flowing in the manometer is kept in connection with both the pleural cavity and the gas bottles, and registers, in practice, the pressure in the latter. This pressure, the pressure at which gas is being entered, is kept at any desired figure by an adjustment, by means of blocks or other apparatus, of the heights of the columns of fluid in the two bottles. By cutting off the gas bottle by finger pressure the manometer can be made, at any moment, to record the intrapleural pressure, and when this rises to the level of the pressure in the bottles, and the flow of gas stops, the pressure bottle must be again raised if more gas is required to be put in.

Removing the Needle.—When the desired amount of gas and pressure is reached, the clamps are replaced on the tubing of the bottle system, the manometer reading is recorded, and the pneumothorax needle withdrawn. In extracting the needle it is advisable to lift up a large fold of the chest wall with it, and after its withdrawal, by deep manipulation and rubbing of this to obliterate the needle track as far as possible, so as to prevent the occurrence of surgical emphysema. No dressing is needed for the small puncture wound. The patient will probably be quite comfortable after the operation, and will very likely be drowsy from the effects of the

morphine. If there is any pain or discomfort a light poultice will, as a rule, readily relieve it.

Effect on Chest Signs.—The effect on the physical signs of a 300 c.c. first injection is usually remarkably small, and is generally more noticeable some hours later than immediately after the operation. An area of increased resonance of varying extent, and some muffling of the breath sounds and râles, are very commonly demonstrable about the spot where the gas was injected, and this is particularly interesting when it is borne in mind that the old belief in “cohesion” of pleural surfaces is said to be definitely disproved. Indeed Emerson has shown by experiments on dogs that a small amount of gas introduced into the healthy pleural cavity always finds its way to the highest point in whatever position the body is placed. In the presence of disease of lung and pleura, such as occurs in artificial pneumothorax cases, this impartial wandering of gas probably takes place to a much smaller extent.

Hints for First Operation.—Not to put in more than 300 c.c. of gas as a rule.

Not to raise pressure by more than 10 cm. H_2O .

Not to attempt more than two punctures at first operation, as thereby the risk of pleural reflex is materially lowered. In this connection Saugman recommends two punctures at a first sitting, and, if these fail, three or four attempts at a second sitting, if the patient is not over-nervous. He remarks that an efficient pneumothorax is generally found at the first puncture, rarely at the second to the fourth, and never after this, though in his earlier practice he was accustomed to puncture up to twenty or thirty times.

DIFFICULTIES ATTENDING INITIAL OPERATION

In the foregoing description it was presumed, for convenience, that the puncture discovered a free pleural space, as evidenced by a characteristic manometer reading, and nothing remained but the introduction of

gas. Unfortunately things do not always proceed so smoothly as this, and certain possible variations from the optimum must receive consideration.

(1) **Absence of Manometric Response.**—The characteristic “snap” which generally announces the passage of the needle through the parietal pleura may occur, but the expected manometer fluctuation fail to follow. This may indicate closely adherent pleura, or it may be due to a blocking of the needle with blood or other fluid, or with solid tissue. The latter possibility is readily excluded by passing the stilette through the needle, the proper use of the stuffing box not being neglected. If this fails adherent pleura may be presumed, but in some cases there will remain a doubt as to whether the pleura has really been passed, and the cannula may then be cautiously pushed forward to see what further obstruction meets it. No force must be used or an adherent lung may be torn—the educated touch can generally appreciate the inelastic resistance of adherent lung, and the blunt cannula end will not penetrate this (unless a soft caseous patch is encountered) if proper gentleness is used. Those who use, the writer thinks unwisely, a pointed needle for the first operation, will doubtless pass it onward into the lung. In this case two possibilities are open to them:

(a) They may enter the lung parenchyma or a bronchial twig, when manometer fluctuations of 3 or 4 cm. H_2O round the atmospheric pressure, or only a trifle below this, will occur. Saugman says that the fluctuations with the end of the needle in the lung may range as large as -6 to $+4$ cm., and, rarely, if the portion of lung is shut off from a bronchus, a negative pressure as high as -3 or -4 cm. mean pressure may be observed.

(b) The needle may perforate a vessel, when a small fluctuation occurs, but soon stops from clotting in the needle and the resistance of capillarity. In the case of a vein, blood may not enter the needle but gas may be sucked into the vessel (see Gas Embolism).

(2) **Incomplete Manometric Response.**—When adhesions are present and are not very dense, the manometer may register a fall of pressure of a few centimetres accompanied by some amount of respiratory fluctuation. As a rule the fluctuations will be irregular in extent and vary with slight movements of the needle. Under these conditions no clear evidence of being in the pleural cavity exists, and the needle is withdrawn and entered at another spot.

(3) **Negative Pressure without Respiratory Fluctuations.**—After apparent perforation of the parietal pleura, a large negative fluctuation (10 cm. or so) of the manometer fluid occurs, and this then remains fixed. So large a drop of pressure is proof that the end of the needle is in the pleural space and the lumen of the needle may have become blocked, but a similar phenomenon seems to occur in the presence of loose adhesions among which the manometer gas becomes lost and is not returned. Under these circumstances the writer has been accustomed to withdraw the needle and try again near the same spot or elsewhere. We have it on the authority of Saugman that with a negative pressure of 12 to 15 cm. H_2O the certainty of being in the pleural cavity is absolute, and gas may be safely run in although the fluctuation is in abeyance. The writer would object to this that when adhesions are present it is difficult to keep the needle point in the pleural space, and though it may have been there at the moment of the manometer fluctuation, evidence that it is still there at the moment the gas should be entered is, in the absence of manometer fluctuations, altogether wanting.

(4) **Pleural Pockets.**—On passing the parietal pleura the usual manometer fluctuation occurs, but 60 or 70 c.c. of gas suffice to raise the pressure above the atmospheric with but weak respiratory fluctuations, showing that a pleural pocket has been entered. The pressure should now be raised to + 5 cm. H_2O or thereabouts, and if there is any outlet it will fall again

gradually and more gas can be entered. If the pressure does not fall and respiratory fluctuations continue, a further pressure up to 10 or 15 cm. H_2O (Saugman) may be tried, but no more at a first operation. At subsequent fillings the pressure may be raised to 20 or 25 cm., and, in many cases, more in an attempt to obtain an efficient pneumothorax (see p. 79). If the pocket found seems to offer small hope of success, the needle may be withdrawn and search made for a larger pleural space to work on or, failing its discovery, two or more pockets may be enlarged simultaneously with a view to uniting them, after the manner recommended by Brauns. It has been asserted by v. Muralt that the intrapleural pressure, taken with the breath held, affords useful information of the capacity of the free pleural space entered, this showing a negative pressure of -10 to -4 according to the size of the pocket. This seems to the writer a very doubtful proposition, since among his cases the negative pressures in quite small pockets were as high as those where a complete pneumothorax was subsequently attained. Even in a pocket too small to admit a measurable quantity of gas the writer has obtained a fluctuation of -13 to -6 , and this seems to be confirmed by the experience of Saugman. The following initial pressures among cases of the writer's serve to exemplify this point:—

Initial Pressure in Cases of Complete Pneumothorax.	Partial Pneumothorax Cases. Efficient.	Partial Pneumothorax Cases. Inefficient.
— 16 — 8 cm. H_2O .	— 12 — 4 cm. H_2O .	— 13 — 9 cm. H_2O .
— 12 — 8 „ „	— 15 — 7 „ „	— 12 — 8 „ „
— 14 — 1 „ „		— 16 — 9 „ „
— 13 — 4 „ „		— 18 — 12 „ „

(5) **Puncture of the abdominal cavity** through an unexpectedly high diaphragm, a rare and in most cases avoidable accident, shows a negative pressure with reversed respiratory fluctuations. In some cases, however, the fluctuations are said to follow the normal type at first, and only become paradoxical after some gas has been allowed to enter.

CHAPTER V

EFFECT OF PLEURAL ADHESIONS: PARTIAL AND COMPLETE PNEUMOTHORAX

PLEURAL adhesions are the rock on which the immediate success of pneumothorax therapy is most commonly wrecked, for without a sufficiency of free pleural space an efficient pneumothorax cannot be produced. Adhesions entirely prevented the production of a pneumothorax in 11% of Saugman's cases, in 24% of Zink's, in 15% of Hamman and Sloan's, in 12.5% of Keller's, in 7.5% of Weiss's, in 9.1% of Brauns', and in 20% of the writer's cases up to date. Among 757 American cases collected by Sachs, failure was recorded in 21.5%. But even where a clear space can be found and gas be introduced, adhesions may prevent sufficient collapse of the lung to render the pneumothorax therapeutically useful. Such a result sufficed to swell the figure for adherent pleura to 27% of Zink's cases, 22% of Saugman's, 37.5% of Keller's and 11.2% of Braun's, and 37% of the writer's. The marked discrepancies of these figures is due doubtless to the different class of cases selected for treatment by these several physicians—thus the high percentage of adherent pleura among the writer's material is certainly due to the fact that he has been content to confine this treatment for the most part to cases of advanced "unilateral" disease, for the farther advanced the disease the greater the likelihood of widespread adhesions.

The common presence of adhesions, to a greater or less extent, among cases submitted to pneumothorax treatment, and the mechanical effects due to their presence,

dictates a natural division of pneumothorax cases into three fairly well-marked clinical groups.

PARTIAL PNEUMOTHORAX

Ineffectual.—These are cases where a small pneumothorax is produced, often with difficulty, and all subsequent attempts to enlarge it to a useful extent fail. It is often difficult to prophesy at the outset how far a partial pneumothorax may benefit a patient. The writer has seen arrest and cure of disease produced by gas collections filling less than one-half of the pleural cavity. It can never be determined beforehand, either, whether a pocket of gas can or cannot, under suitable pressure, be expanded gradually into an efficient pneumothorax. Experiment is, in both cases, the only reliable criterion. Where, however, to give a gross example, in a patient with extensive cavitation only a small and remote gas pocket can be achieved, and where a few fillings at 35 to 40 cm. H_2O pressure fail to exert any influence on it, it soon becomes obvious that it is useless to proceed. Examples of partial pneumothorax which refused to yield to pressure and were abandoned as ineffectual are here given.

H. B., aged 34 years.—Under my care on different occasions since 1905, with attacks of hæmoptysis up to 2 pints on some occasions. Moist sounds over the whole left side, but no cavity signs and no certain cavity visible to X-rays. Sputum 2 or 3 oz. daily. General condition good. No less than seven attempts to find free pleura were made on three occasions and failed, but at the eighth, on 16.2.15, a free pocket was found at the base behind and 350 c.c. of N put in.

16.2.15 . — 16 — 9 cm. H_2O .	350 c.c.	+ 4 + 12 cm. H_2O , falling in a few minutes to — 4 + 4 cm. H_2O .
17.2.15 . — 10 — 1 „ „	150 c.c.	+ 17 + 20 „ „ soon falling to + 15 + 18 cm. H_2O .
19.2.15 . — 9 — 1 „ „	100 c.c.	+ 33 + 36 „ „ soon falling to + 29 + 32 cm. H_2O .

A pocket at the base rising about a quarter to one-third of the way up the back was thus formed, and gas was put in at a pressure just under + 40 every week, later every two, and finally every three weeks, the pressure on each occasion slowly but steadily falling from a leakage of gas inside the chest, presumably among the adhesions. The amount of gas was generally about 300 c.c., and this could be increased to 400 c.c. or so by waiting for it to spread itself, thus :—

21.12.15	.	.	— 9 — 4	300 c.c.	+ 35 + 37 falling
11.1.16	.	.	— 10 — 4	350 c.c.	+ 30 + 34 „
1.2.16	.	.	— 8 — 3	325 c.c.	+ 33 + 37 „
22.2.16	.	.	— 9 — 2	400 c.c.	+ 34 + 38 „

The hæmorrhages were somewhat controlled but not abolished, and treatment was persisted with for nearly eighteen months at the patient's desire, and with a view to testing the possibility in such a case of achieving success by persistent attack. The pneumothorax cavity, however, showed no increase in size, whether to X-rays (see Plate I, facing this page) or to physical examination; it was but doubtfully effective, and has now been finally abandoned.

G. M., aged 25 years.—Chronic disease of whole right side, with cavitation below the apex and moist sounds to base; left side practically free of signs. T. B. + Heart pulled over to right.

[9.5.16	.	At second puncture free pleural space found at fifth space in mid axilla.			
		— 18 — 12 cm. H ₂ O.	300 c.c.	+ 6 + 8 cm. H ₂ O.	rapidly falling
12.5.16	.	— 8 — 4 „ „	350 c.c.	+ 15 + 18 cm. H ₂ O.	soon falling to
				+ 8 + 10 cm. H ₂ O.	
16.5.16	.	— 8 — 4 „ „	225 c.c.	+ 20 + 21 „ „	falling
19.5.16	.	— 4 to 0 „ „	200 c.c.	+ 26 + 28 cm. H ₂ O,	falling
23.5.16	.	— 4 to 0 „ „	200 c.c.	+ 34 + 36 cm. H ₂ O,	falling
30.5.16	.	— 3 + 1 „ „	200 c.c.	+ 28 + 29 cm. H ₂ O,	falling

Pleural pain was experienced near the end of each earlier filling, generally at the base, but on one occasion conducted to the shoulder region. Only a very small basal pneumothorax was formed, and as it yielded little to pressure and could obviously exert no important influence on the extensive cavitation near the top of the lung, the treatment was abandoned.

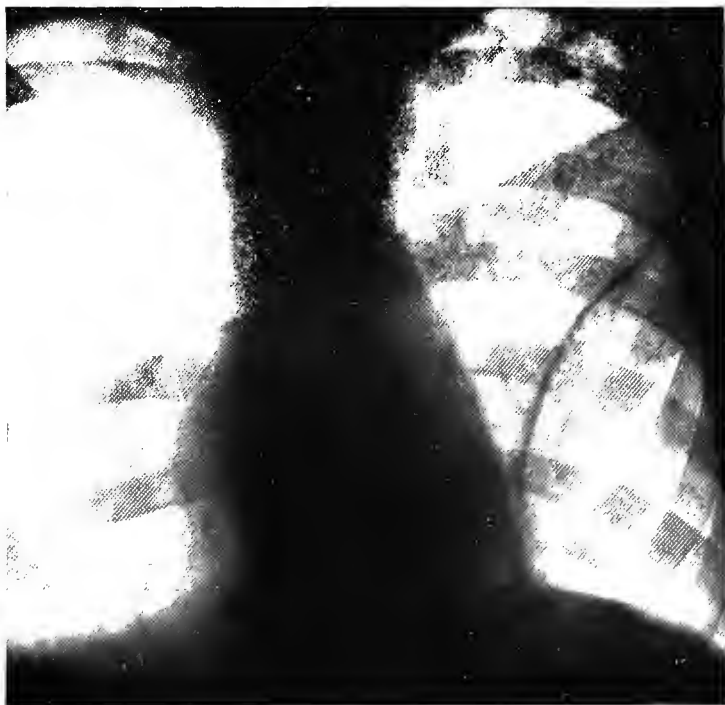


PLATE I.—RADIOGRAM OF CHEST OF H.B., WHOSE CASE IS DESCRIBED
ON PAGE 53.

It shows a small pneumothorax at the left base, disease in the lung
above, and quiet or arrested disease in the right lung.

Taken Aug. 24, 1916, by Dr. Overend.

Effective.—As already remarked the value of a partial pneumothorax must be decided by practical results. Often an inconsiderable pocket of gas can be enlarged by repeated refills at a gradually rising pressure. Forlanini gives these refills daily until the desired result is achieved. Brauns, in cases where only pleural pockets can be found, puts in gas at several adjacent points, and strives by a rising pressure to unite them. The possibility of enlarging an inefficient pneumothorax till it becomes of therapeutic value may depend not only on the strength of adhesions to be separated, but also on the resistance of surrounding structures to the needful pressure. In particular the mediastinum must be carefully watched since its weakness may lead to displacement or bulgings which may set a clear limit to the amount of pressure which it is safe to employ. (See Mediastinum, pp. 105 and 106.) This difficulty does not often arise where the gas collection is still of small size. Adhesions may either separate or stretch, and to stretching the diaphragmatic pleural bands are said to be most amenable, and to give rise but seldom to difficulty. An example of a small pneumothorax enlarging under pressure till it became fully effective will here be given.

H. B., aged 34 years.—Five months' history of dyspnœa, cough with expectoration, and loss of weight. Nutrition poor, sputum one ounce daily. T.B. +. Moist crepitations over the whole left side and extensive excavation about the middle of the lung giving typical cavity signs in front, axilla, and near spine. Right side practically clear.

23.3.14 . At second puncture free pleura found below the scapular angle.					
	. — 12 — 4 cm. H ₂ O.	250 c.c.	—	4 + 2 cm. H ₂ O.	
25.3.14 .	— 7 to 0 „ „	450 c.c.	+	4 + 9 „ „	
27.3.14 .	0 + 2 „ „	350 c.c.	+	9 + 11 „ „	
31.3.14 .	+ 3 + 7 „ „	350 c.c.	+	12 + 14 „ „	
7.4.14 .	— 1 + 1 „ „	600 c.c.	+	14 + 15 „ „	
17.4.14 .	0 + 7 „ „	600 c.c.	+	16 + 20 „ „	

A small pneumothorax was formed at the base extending up to the fifth rib in front and about one-third up the back. Pressures up to about 20 cm. H₂O were continued at each filling and in about three months the gas chamber extended up to the

third rib, cavity signs were abolished, and crepitations had become inaudible even at the apex. Expectoration had practically ceased, and weight was rising. The heart was displaced to the right nipple line, but the patient experienced no inconvenience, and returned to his work as a cabinet maker about the middle of May 1914. The fillings were now larger.

30.6.14	.	-	1 + 4	.	.	975 c.c.	+	19 + 21
21.7.14	.	-	5 + 1	.	.	1,100 c.c.	+	19 + 21
7.8.14	.	-	2 + 2	.	.	1,075 c.c.	+	20 + 22
28.8.14	.	.	0 + 5	.	.	900 c.c.	+	15 + 17

He kept in good health, the functioning lung remained clear of signs, as did also the adherent upper part of the collapsed lung. In the spring of 1915 he had a small pleural effusion which lasted about two months, and was then absorbed; it did not in any way put him back, but diminished gas absorption to a convenient extent, so that he was able to retain his gas over a longer interval. He continues his work and returns every month for a refill as :—

4.1.16	.	-	10 - 6	.	.	525 c.c.	+	17 + 19
8.2.16	.	-	7 - 2	.	.	400 c.c.	+	18 + 21
10.3.16	.	-	7 - 3	.	.	300 c.c.	+	18 + 21
11.4.16	.	-	9 - 5	.	.	450 c.c.	+	17 + 20
9.5.16	.	-	8 - 4	.	.	450 c.c.	+	18 + 20

COMPLETE PNEUMOTHORAX

This term must be used in not too limited a sense, since it is convenient to include under it those cases, all too common, where adhesions exist, often at the apex, without detracting from the clinical completeness of the result. Where, however, there is cavitation at the apex and the whole apical cupola is adherent, this may prevent or seriously impede the emptying and obliteration of cavities, and the case will fall into a different category. A complete pneumothorax is one where the lung as a whole is able to attain an efficient collapse, and a few adhesions, providing they are not too far from its root, may not sensibly interfere with this result. The earlier in their course cases are submitted to treatment the more often will a complete pneumothorax be obtainable; nevertheless, with advanced one-sided disease it not infrequently happens that a fairly complete pneumo-

thorax is attained, even though this may occur at a fairly late stage of the treatment. An example of complete pneumothorax in a case of advanced disease will be here given with outline notes covering a year's treatment.

W. N., aged 24 years.—A history of cough and wasting over many years. In-patient at City of London Hospital for Diseases of the Chest six years ago, and then had tubercle bacilli in the sputum. Rather thin, hoarse with tuberculous ulceration of the larynx, some dyspnoea. Pulse 108: Temp. 99° – 102.2° : Resp. 24. Chest emphysematous. Moist crepitations all over right lung to base: good tidal movement at base. Left lung practically clear. Heart, dulness wide, from L.N.L. to $2\frac{1}{2}$ inches to right of mid-sternal line. A radiogram showed disease of both lungs, on the right side large blotches of shadow throughout the lung, but most marked in the upper two-thirds; on the left side smaller shadows extending out into two-thirds of the lung mostly towards its middle, and with a tendency to coalesce into larger masses near the root.

14.5.15 .	Free pleura found at first puncture in fourth space close to nipple.		
	— 13 – 4 cm. H_2O .	300 c.c.	— 10 + 2 cm. H_2O
18.5.15 .	— 12 + 1 „ „	500 c.c.	— 8 + 4 „ „
21.5.15 .	— 10 to 0 „ „	550 c.c.	— 5 + 7 „ „

By this time breath sounds feeble all over right side and crepitations abolished. Heart $\frac{3}{4}$ inch outside L.N.L. Temperature here fell to normal and has remained so since (see Fig. 4, p. 58).

25.5.15 .	— 10 + 2 . .	800 c.c.	— 4 + 7
-----------	--------------	----------	---------

Right side breath sounds nearly abolished; percussion note high pitched. V.R. and V.V. unaltered. Left side a few crepitations in lower half. Heart 1 inch outside L.N.L.

27.5.15—*X-ray screening.*—Diaphragm moves freely; inspiratory suction of mediastinum to pneumothorax side. Lung dense and of largish size, lies against mediastinum and shows no respiratory movements. Unimportant adhesions at apex and base.

28.5.15 .	— 6 + 1 . .	500 c.c.	— 3 + 6
1.6.15 .	— 2 + 2 . .	275 c.c.	— 1 + 5
8.6.15 .	— 4 + 2 . .	625 c.c.	+ 1 + 8

Left lung now free of crepitations.

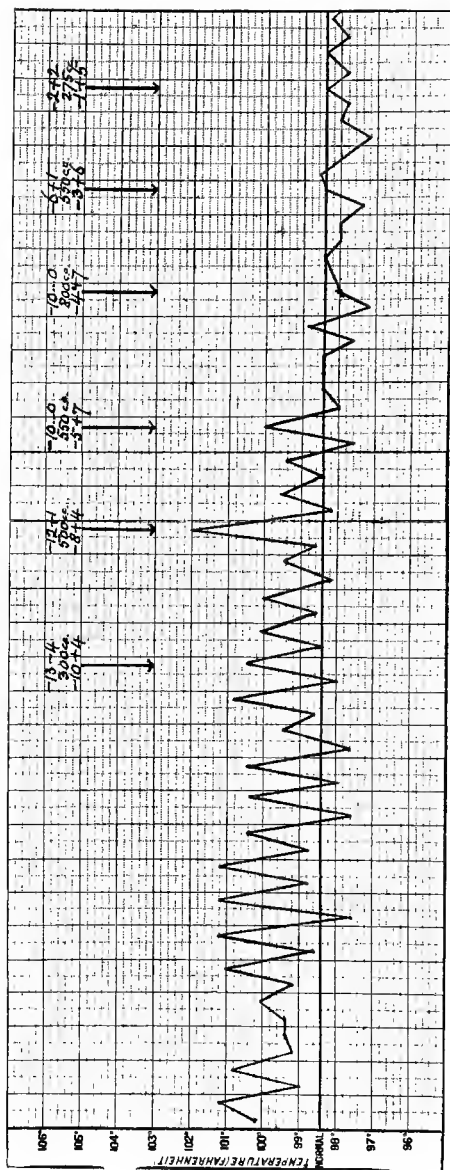


FIG. 4.—TEMPERATURE CHART OF W.H., SHOWING THE EFFECTS OF PNEUMOTHORAX ON THE FEVER.



PLATE II.—RADIOGRAM OF CHEST OF W.N., WHOSE CASE IS DESCRIBED
ON PAGE 57.

It shows the collapsed right lung lying against the mediastinum, a strong adhesion at its base; also areas of disease in the functioning lung.

Taken on Oct. 28, 1915, by Dr. Overend.

16.6.15	. - 4 + 2	. .	600 c.c.	+ 2 + 8
22.6.15	. - 1 + 4	. .	450 c.c.	+ 4 + 7
29.6.15	. - 2 + 6	. .	350 c.c.	+ 1 + 10
9.7.15	. - 3 + 5	. .	400 c.c.	+ 1 + 9

Heart 1 inch outside L.N.L. and 2 inches to right of mid-sternal line. A few crepitations outside heart. Percussion note somewhat high pitched both sides of chest.

17.7.15	. 0 + 4	. .	275 c.c.	+ 2 + 10
27.7.15	Feels a "splash" when he moves. A little fluid found.			
	0 + 5	. .	240 c.c.	+ 2 + 9
3.8.15	Fluid now 2 inches up.			

10.8.15	. Less fluid.			
	- 2 + 4	. .	300 c.c.	+ 2 + 9

24.8.15	. Now only 1 inch of fluid.			
	- 6 to 0	. .	570 c.c.	+ 3 + 10

3.9.15 . Fluid gone. It was ushered in with no symptoms and no fever, but temp. rose to 99°-99.5° in evening between 3.8.15 and 13.8.15.

	- 4 + 1	. .	425 c.c.	+ 2 + 9
14.9.15	. - 4 + 3	. .	450 c.c.	+ 6 + 10
24.9.15	. - 3 + 5	. .	300 c.c.	+ 6 + 10
12.10.15	. - 1 + 6	. .	150 c.c.	+ 1 + 8

Radiogram on 28.10.15 shows the shadows in the left lung are smaller and denser than at beginning of treatment five months ago, and tend to range themselves into a more reticulated outline (see Plate II, facing this page).

9.11.15	. - 6 + 4	. .	250 c.c.	0 + 7
---------	-----------	-----	----------	-------

By now has gained 12 lb. in weight.

7.12.15	. - 8 to 0	. .	350 c.c.	- 1 + 9
5.1.16	. - 10 + 1	. .	400 c.c.	+ 1 + 10

1.2.16 . Has now gained 15 lb. in weight since beginning of treatment. Left lung quite clear to physical examination.

	- 9 - 1	. .	420 c.c.	+ 4 + 11
--	---------	-----	----------	----------

Left Hospital on 2.2.16 and returned for refills.

29.2.16	Measurement R. chest, 17 $\frac{3}{4}$ in.			
		L. „	16 $\frac{1}{2}$ in.	
	- 9 - 1	. .	300 c.c.	+ 1 + 7
28.3.16	. - 12 to 0	. .	360 c.c.	+ 1 + 7
2.5.16	. - 11 - 1	. .	450 c.c.	+ 2 + 10
30.5.16	. - 8 + 2	. .	250 c.c.	- 1 + 7

This gives an outline of gas fillings and pressures during the first year of treatment in an average case of complete pneumothorax.

The patient left the Hospital early in February 1916, and has since lost some weight, but seems fairly well, though some sputum remains, doubtless from central areas in the functioning lung. This lung remains clear of stethoscopic signs, and has a tidal excursion of $\frac{1}{2}$ inch on the posterior aspect. On account of his emphysema and some consequent cardiac embarrassment he is always decidedly dyspnoëic and complains of an increase of this "tightness" after his refills.

DIAGNOSIS OF ADHERENT PLEURA

In recommending the adoption of pneumothorax treatment in any individual case, it is right that some attempt should be made beforehand to gauge the likelihood of finding a free pleural space. Though this is notoriously a matter of difficulty in some cases, such points as are likely to be helpful in making the decision will here be outlined.

In the first place, the earlier and the more localised the disease the less likely are widespread adhesions present. For this reason, among others, useless delay must be avoided as soon as it is clear in any case that other methods have failed to arrest the spread of disease, and that no clear contraindications to pneumothorax therapy exist. The decision must be prompt and must be put to early trial. On the other hand, disease involving the whole of one lung by no means predicates adherent pleura of such extent that a useful pneumothorax cannot be obtained. The writer has produced a practically complete pneumothorax in many cases where crepitations extended from apex to base, and also in cases of predominantly basal disease.

A clear history of pleurisy (not merely dependent on the patient's statement) certainly gives some likelihood of adherent pleura, but it must be noted that pleurisy by no means always leads to adhesion of the pleural surfaces. Adherent pleura may announce itself by dragging, or interrupted breath sounds, or by a local shortening of inspiration. Particularly suspicious are superficial creaking sounds over a lung extensively

diseased. Feebleness of breath sounds at the base is of ominous significance—particularly is this the case if marked displacement of organs and evidence of fibrosis are present. In these cases it must be noted that even if a pneumothorax is capable of production, the capacity of a chronically diseased and fibrosed lung to collapse must be somewhat limited. Nevertheless the writer has often in practice obtained quite useful collapse in lungs of this description. Large superficial cavities are nearly always covered by extensive and dense pleural adhesions, and in many, perhaps most, these adhesions are incapable of separation, and the cavity thus incapable of complete extinction under pneumothorax therapy. On the other hand the writer has seen cases where the thickening involved the visceral pleura only, and but few or no adhesions were present to bind the lung to the chest wall. In the writer's experience troublesome adhesions have been found more commonly on the left than on the right side of the chest. The most reliable test of free pleura exists in the tidal movement of the lower edge of the lung during inspiration and expiration (see p. 40); if a clean ample excursion is noted it is practically certain that a useful pneumothorax can be achieved. On the other hand, if movement is restricted or abolished, it does not necessarily follow that adhesions are present at the base—such a limitation may be due to adhesions higher up the side or to marked pulmonary changes leading to diminished expansile power of the lung itself. Finally the use of the X-rays may help to decide the presence or absence of adhesions—the finely stippled shadows of pleural thickening may be noted, or a basal pleurisy may lead to distortion and restricted movement of the diaphragm.

THE REDUCTION OF ADHERENT PLEURA

In cases where no free pleural space can be found, an attempt to create one is recommended by some

operators. Forlanini's method is to enter his needle just short of the pleural space and then to move it slowly forward, at each movement forcing out some $\frac{1}{2}$ c.c. of gas by pressure of the fingers on the manometer tubing. If the gas thus pressed out enters the tissue outside the pleura, the manometer shows a negative fluctuation equal to the volume of gas forced in; if the gas bubble is very near the pleura there may be small oscillations. Where the gas gets between the pleural layers and separates them more marked negative pressure and wider respiratory fluctuations occur. When these show themselves an attempt is made to enter more gas under pressure—if the needle is outside the pleura still the rising pressure gradually falls again as the gas diffuses through the tissues. If the needle misses the pleural cavity and enters the lung, small oscillations about the zero point occur. Saugman's plan is to pass the needle into the lung and then cautiously withdraw it till oscillations cease. He then uses his suction syringe to exclude a wounded vein, and if no blood comes he forces in Nitrogen under a pressure of 20 to 30 cm. H_2O . If the pleural surfaces become separated a negative pressure will occur after some cubic centimetres of gas have entered. More may then be put in if pain allows.

These operations cannot be considered entirely free of danger even in the hands of experts, and must generally fail to achieve their object—namely, the production of an efficient pneumothorax. They should only be attempted, if at all, in cases where pneumothorax offers a last chance but a hopeful one, and where the patient understands the immediate risk and elects to face it.

If these operations fail or are not attempted, and collapse of the lung is urgently indicated, there remains as an alternative the trial of some form of thoracoplastic operation. Of these the one which appears to offer most chance of success is the "Pfeilerresektion" devised by Wilms. Portions of the posterior

ends of the upper seven to nine ribs are removed at a first operation, and subsequently, after an interval, portions of the costal cartilages of the first five or six ribs through an anterior incision. By this means the ribs are "mobilised," and losing their support drop downwards and inwards, leading to a very complete collapse of the side of the chest and with it the contained lung, especially in its upper parts.

CHAPTER VI

REFILLS

INTERVALS AT EARLY STAGE

HAVING successfully initiated a pneumothorax the question of enlarging and maintaining this needs consideration. It has been calculated that the healthy pleura will absorb some 100 c.c. or more of nitrogen daily, though in conditions of disease absorption is somewhat slower. Balboni after putting in 400 c.c. at an initial operation discovered gas still present at the end of a month. In practice it is found that absorption is rapid at first and tends gradually to diminish, so that the refills begun at short intervals will be needed at longer and longer spacing even up to two months or more in some cases. It is convenient to give the first refill the day after the initial operation, or, at latest, on the second day. An extra day or two can then be added to each succeeding interval until collapse is as complete as can be obtained; if marked autoinoculation follows any of these fillings it may be wiser under certain conditions (see p. 89) to postpone the next refill for a day or two. Later the interval will be judged for each case according to the rapidity of absorption and the ability of the lung to remain collapsed under a falling pressure.

OPERATION OF REFILLING THE PNEUMOTHORAX

This operation differs in but few respects from the initial operation already described. The main points of distinction are as follows :

(1) No general anodyne is necessary in the case of refills unless in a highly nervous and apprehensive patient for the first one or two.

(2) There is no longer need to avoid the use of a sharp-pointed pneumothorax needle, since there now exists a substantial pocket of gas to protect the lung against any possibility of injury. Suitable needles for use at refills have been already described on p. 36. They must be sterilised in absolute alcohol and dried by heat in similar manner to that used for the trocar and cannula at the initial operation. Care must be taken that the tap of the needle, whereby it would communicate with the outer air, is closed before the operation is commenced.

Puncture Spot.—The spot for puncturing at the first refill is best chosen near the original puncture so as to be well within the gas pocket should the gas be localised by adhesions. In addition it is important at the first refill that the operation spot be directly uppermost so that, in a free pleural cavity, the maximum of gas may collect here and protect the lung from puncture. The position of the gas can generally be pretty accurately defined by gentle percussion if any doubt exists as to its location. At later refills, when the pneumothorax has become complete or at any rate large, the puncture may be made at some other part of the chest if more convenient. A change of position may be dictated by progressive thickening of the pleura at the spot where repeated punctures have been made. In puncturing in a new neighbourhood due caution must be directed to the possible presence of stretched adhesions, which in some cases may contain lung tissue. These provide the possibilities both of gas embolism and of infection of the pleural cavity if a piece of lung is pierced, accidents which otherwise should be well out of reach in the course of an ordinary refill. Novocaine should be used, as at the initial operation, since the risk of pleural shock cannot be entirely put aside.

MANOMETER READINGS

Intrapleural Pressure.—The needle is entered slowly and steadily with the eyes closely fixed upon the manometer to catch the moment when the needle end enters the pleural cavity; when the pneumothorax is large the needle may be pushed well in so as to support its own weight. The manometer will not necessarily, as at the first operation, register a large negative pressure, but the reading will depend on many factors, and in particular, on the final gas pressure at the preceding refill, and the time which has since elapsed. At the first or second refill a considerable negative pressure is still to be expected in the case of a complete or nearly complete pneumothorax. Thus, for example:

W. M.

2.2.14 .	Initial Operation	— 16 — 8 cm.	325 c.c. — 5 to 0 cm.
3.2.14 .	. . .	— 12 — 4 „	700 c.c. — 2 + 5 „
6.2.14 .	. . .	— 5 + 1 „	950 c.c. + 3 + 10 „

R. J.

9.3.14 .	Initial Operation	— 12 — 8 cm.	300 c.c. — 1 + 1 cm.
10.3.14 .	. . .	— 8 — 5 „	700 c.c. + 8 + 10 „
12.3.14 .	. . .	— 7 — 3 „	600 c.c. + 11 + 13 „

Where only a partial pneumothorax has been produced the fall of pressure is much less striking. Thus:

H. B.

23.3.14 .	Initial Operation	— 12 — 4 cm.	250 c.c. — 4 + 2 cm.
25.3.14 .	. . .	— 7 to 0 „	450 c.c. + 4 + 9 „
27.3.14 .	. . .	0 to +2 „	350 c.c. + 9 + 11 „

At later refills the fluctuations are commonly round about the atmospheric line at the time the refill is usually required; in some cases more below than above, but commonly more above than below, and where with a well-collapsed lung absorption is rendered slow by the presence of thickened pleura, the pressure

may remain positive even at the end of very long intervals. In these results the condition of the lung undoubtedly exercises a decided influence. Where the lung is well compressed, absorption is slow and a positive pressure is readily maintained; where, on the other hand, the lung is imperfectly collapsed the pressures quickly fall to or below the atmospheric, and larger quantities of gas are needed to maintain the pneumothorax. In theory, of course, the latter class of case should be eliminated by the production of a more efficient pressure, but in practice the class exists just because this cannot be done, the usual bar being a discrepancy between the resistance of the lung to compression and of the mediastinum to displacement. A few examples of these different conditions of pressure are here given.

H. W. Pressure inclining to negative :

21.7.15 .	13th Filling	- 4 + 2 cm.	650 c.c. + 6 + 10 cm.
3.8.15 .	.	- 6 + 3 „	825 c.c. + 4 + 11 „
17.8.15 .	.	- 5 + 3 „	650 c.c. + 2 + 11 „
31.8.15 .	.	- 7 + 4 „	740 c.c. + 3 + 11 etc.

W. N.—Pressure inclining to positive :

22.6.15 .	9th Filling	- 1 + 4 cm.	450 c.c. + 4 + 7 cm.
29.6.15 .	.	- 2 + 6 „	350 c.c. + 1 + 10 „
9.7.15 .	.	- 3 + 5 „	400 c.c. + 1 + 9 „
17.7.15 .	.	0 to + 4 „	275 c.c. + 2 + 10 etc.

M. P.—Pressure wholly positive :

10.11.14 .	.	+ 2 + 8 cm.	175 c.c. + 16 + 21 cm.
22.12.14 .	.	+ 4 + 10 „	150 c.c. + 16 + 21 „
5.1.15 .	.	+ 2 + 8 „	175 c.c. + 16 + 21 „
23.3.15 .	.	+ 1 + 7 „	200 c.c. + 17 + 21 etc.

This last was a case of partial pneumothorax of old standing with a thickened pleura, and absorption was reduced to a minimum.

The most disturbing condition to encounter is one with small fluctuations about the atmospheric—a manometer reading very similar to that which would be

obtained with the needle thrust into a bronchus or the lung parenchyma. Thus :

E. H. B.

17.11.14 . . . - 4 + 4 250 c.c. + 11 + 17

and in the same patient, a case of partial pneumothorax, at a later stage—

29.6.15 . . . - 3 + 3 175 c.c. + 21 + 25

These cases are disturbing because the possibility of the needle passing into or through adhesions has always to be kept in mind. It is, indeed, a fact that most of the recorded cases of fatal gas embolism have occurred during refills and not at the initial operation. Scrupulous care must therefore be exercised at refills, no less than at the first operation, to make certain that the needle is in the pleural space before gas is entered. For this, the wide, easy, respiratory fluctuations of the manometer fluid are usually a sufficient guarantee, and this evidence may be safely supplemented, in the writer's opinion, by the manometric effect of a gentle short cough. No deep breath must be taken, and the cough must be gentle indeed or the fluid may be driven out of the manometer tubing. This accident has often to be guarded against during refills by getting the patient to give notice of the need to cough and by firmly compressing the tubing between the needle and apparatus till the coughing is finished. If the characteristic clear swing of the manometer fluid fails at a refill, no gas must be entered until this swing has been established or re-established. Its absence often implies a blocking of the needle, but this must not be presumed without proof, and unless the needle can be cleared, it must be withdrawn, re-sterilised and dried, and entered elsewhere. Fortunately needles of over 1 mm. calibre, such as the Lillingston or Saugman needles recommended, very

rarely get blocked if used with proper care, but the small needle below 1 mm. of the type used by Forlanini, Brauns and others readily becomes obstructed or partially obstructed, generally by a piece of punched-out skin, so that a clean manometer swing fails to appear. When this occurs the temptation to presume a blocked needle is so great that the writer has himself entirely discarded the use of needles of this calibre as likely to lure the operator into unjustifiable risks.

Finally, at a refill, it is always open to the operator to prove the intrapleural location of the needle point in cases of doubt by the withdrawal of some gas from the pleural cavity into the gas bottle. This is achieved by lowering the pressure bottle and must be followed, after the conclusion of the refill, by emptying of the gas bottle and sterilisation of the tubing and filter between this and the needle. The writer has never yet resorted to this expedient.

Before leaving the subject of the manometer at refills, a few additional words may be added as to the significance and interpretation of various manometric readings.

Respiratory Fluctuations.—These in their amplitude are dependent on many factors, such as the calibre of the manometer tubing and needle (p. 29), the type of the patient's breathing, as from nervousness, dyspnoea, mouth or nose breathing, and the like. To obtain a uniform manometer reading the patient should be instructed to breathe through the mouth; chest breathing also supplies a notably larger fluctuation than does abdominal breathing, but the patient cannot with advantage be instructed on this point. In addition it will often be found that respiration naturally runs in cycles about a somewhat different mean pressure according to whether inspiratory or expiratory stimuli are in the ascendancy. The respiratory fluctuations also change in the individual case according to the stage of the pneumothorax and the pressure at which it is kept. Thus when gas is first put in the fluctua-

tions are those of the normal intrapleural pressure and are large. At a later stage, when the lung is collapsed, the fluctuations are usually but not always found to become smaller, and several factors seem to the writer responsible for this change. First and foremost is the diminished respiratory movement of the expanded hemithorax released from the elastic suction of the lung. But in addition, factors dependent on the intra-thoracic pressure come into play. Thus with a pressure near the atmospheric the inspiratory change must be to some extent balanced by a movement of the mediastinum towards the pneumothorax side (see p. 105), while the expiratory phase is reduced by its return towards the side of the functioning lung. At higher pressures this mediastinal movement ceases, but then the diaphragm, on whose action the respiratory change of pressure in large part depends, ceases any longer to functionate (see p. 107).

Certain points of practical information are said to be deducible from various types of manometer reading, thus :

Re-expansion of Lung.—If the lung tends to expand in the interval between the fillings there will be a gradual rise of pressure at the refill, whereas if the lung remains collapsed the pressure is said to rise abruptly at the end of the refill (Hamman and Sloan). This has not been the writer's experience.

Condition of Mediastinum.—An elastic flexible mediastinum is shown by a slow rise of pressure, often with wide respiratory fluctuations, whereas a rigid mediastinum causes the pressure to rise rapidly at the end of the refill and the respiratory fluctuations are small (Hamman and Sloan).

These points, it will be seen, are measures of the elasticity of the walls of the gas chamber. The presence of a fluid effusion reduces this, perhaps, to its lowest point, and in this condition the addition or subtraction of a few centimetres of gas may make startling differences in the intrapleural pressure (see p. 137).

PUTTING IN GAS

Having successfully entered the needle the manometer reading is recorded, and connection with the gas bottle established, the level of the fluid being so adjusted that a pressure 5 or 6 cm. above the intrapleural, that is, in most cases a decided positive pressure (above atmospheric) is supplied. The column of fluid in the gas bottle and the patient are carefully watched, as in the first operation, and as soon as some gas has gone in, the pressure bottle may be raised so as to establish a more decided pressure. Gas is passed in, not, as at the first operation, up to a definite quantity; the criterion now and henceforth is one of intrapleural pressure, not quantity of gas.

EARLY FILLINGS. QUANTITY AND PRESSURE OF GAS

In cases where the lung collapses readily, it is wisest to keep below atmospheric pressure till about the third filling of gas (see Case of W. N., p. 57). After this the pressure is gradually raised till collapse is complete. Complete collapse in such cases is generally obtained, if this method is followed, by the fourth or fifth filling, and a sufficient pressure must then be maintained to prevent further expansion. By following this procedure it will be found that some 500–800 c.c. are put in at each filling after the first, perhaps 500 c.c. at the second filling, and 500–800 at the third, fourth, and fifth fillings. It has been calculated that the adult pleural cavity holds some 2,000–3,000 c.c. of gas, but an unknown allowance must be made in each case for reabsorption during the intervals between the refills.

COMPLETE COLLAPSE

What evidence, it will be asked, is needed to decide when complete collapse of the lung has been obtained? The matter can be to a large extent decided by physical signs and symptoms, but the great value of the X-rays in this decision is undeniable.

Radioscopy.—As the volume of gas put in becomes larger the lung is seen to retreat towards the mediastinum, its shadow increasing in density as its size diminishes. Eventually, where a complete collapse is possible, it comes to lie close against the mediastinum in a large gas cavity whose dimensions overstep those of the normal pleural cavity in every direction. For the pleural cupola becomes lifted above the normal lung apex, the mediastinum is bowed over to the healthy side, and the diaphragm assumes a less convex, flattened, sunken, or even in rare cases an altogether inverted position. Seldom, however, is the lung entirely unadherent; more commonly adhesions at apex or base or at points between cause the lung to assume an irregular outline, and to give a shadow of various thickness, size, shape, and localisation on compression. The more effective the compression the deeper and smaller the shadow—only in hepatised lung is a deep shadowing associated with a large but immobilised organ. The test of effective collapse is the failure of the contracted lung to expand with inspiration—mere movements communicated to the lung by respiratory fluctuations of the mediastinum must not be mistaken for changes of volume. In the production of complete collapse stages may be passed through where one lobe of a lung is immobilised but the other still expansile—obviously the elastic, healthy lung tissue is more readily compressed than inelastic diseased parts. On the other hand, once full collapse has been attained, the inelastic diseased part is, as a rule, less easily re-expanded, and a lowering of pressure, whether temporary or continuous, may in some cases be permissible. Saugman describes a case where the lower lobe, the seat of advanced disease, maintained its collapse while the healthy upper lobe expanded by three or four times its volume on deep inspiration. At a higher pressure the whole lung became immobilised. Radioscopy should be applied soon after a filling, and at intervals so that the period during which the lung will remain collapsed,

and the pressure at which it begins to re-expand, may be discovered and thus the intervals best suited for refills decided upon. This may be revised from time to time as the lung, and particularly its most diseased portions, becomes less liable to re-expansion.

Physical Signs.—A general impression of collapse of the lung is obtained where a large or complete pneumothorax has been formed, and the lung has disappeared from view, save, perhaps, to careful percussion in the interscapular region. All moist sounds should have vanished, but breath sounds do not entirely disappear owing to conduction of these from the air tubes or the opposite lung. At the same time movement of the pneumothorax side is diminished, and its girth somewhat increased with a widening of the intercostal spaces. When this condition is reached at a pressure somewhat above the atmospheric (say + 5 mean pressure) there is a presumption that the lung is completely collapsed, but physical signs alone cannot prove the point, and further evidence must be sought from the symptomatology.

Symptoms.—The process of collapsing the lung is accompanied by a squeezing out of secretions—this leads to an increase of cough and sputum after each refill, and when the collapse is complete the sputum disappears or sinks to an even supply. This, in absence of radioscopy, is the best test of the production of collapse, and, if the intervals between the refills are kept short, is a fairly good one. If it is doubtful whether at a given pressure (say 5 cm. H_2O mean) the lung is fully collapsed, a cautious rise to a somewhat higher pressure (say 7 to 10 cm. H_2O mean) a day or two later may decide the matter through the effect or absence of effect of this on cough, sputum, and temperature. In gauging the best intervals between refills the same rough test may be applied. If the interval is so long (and the pressure fallen so low) that the lung re-expands to any marked extent, the subsequent refill will be followed by an increase of cough

and sputum, and possibly by symptoms (fever, headache, etc.) pointing to an autoinoculation. If a still longer interval is left between refills some of the symptoms present before treatment was begun may reappear and gross evidence of re-expansion in fever, cough, wasting, etc., be present. Such a disorganisation of treatment must, of course, never be allowed to occur, and this brings us in more detail to the question of the intervals between the refills.

INTERVALS

It has already been pointed out how, as a routine measure, it is convenient gradually to increase the intervals by a day more each time at the beginning. But when full collapse of the lung has been attained, more rational means for deciding the suitable interval should be adopted. Our purpose is to prevent any re-expansion of the lung, and gas should be put in at such intervals as to prevent this. Signs of re-expansion may be gauged by the means decided above; in addition the rapidity of absorption varies notably with the duration of treatment. Whereas at first the pleural cavity may be expected to absorb some 80–100 c.c. of gas per diem, after the pneumothorax has existed for some months the daily absorption falls to 25–50 c.c. Patients vary somewhat also in the quantity of gas they will tolerate at a refill (*i.e.* the amount they can lose between) without interference with the favourable course of the cure. Forlanini is especially insistent that immobilisation once achieved should thereafter be kept complete. He gives 100–200 c.c. daily until full immobilisation; later every other day, and gradually at longer intervals till after two to four months this gradually suffices to maintain the pressure when given at six to nine days' interval. Even at later stages he keeps the intervals so short that 200–300 c.c. suffices, though in some cases he increases this to 300–500 c.c. These frequent injections and consequent small refills are a

council of perfection which the endurance of the patient and the leisure of the operator may not always permit. At the same time there is no doubt that this is the path of safety, that short intervals and small injections should be aimed at, and that in cases where patients do not improve as they ought with a somewhat coarser procedure, this method should at once be resorted to. It is a very common mistake, in the writer's experience, to leave intervals so long that injections of 800 or 1,000 c.c. are needed to restore the pressure, and this must lead not only to general intrathoracic disturbance from marked changes of pressure, but also in nearly all cases to a notable expansion of the collapsed lung. Where large quantities are needed at short or moderate intervals, this is probably due to an incomplete collapse of the lung, and is an indication for a somewhat higher pressure. 500 or 600 c.c. should probably be the outside quantity of gas at refills, and if this is exceeded the intervals must be correspondingly shortened. Saugman mentions cases where even refills of 400-500 c.c. were too large and led to loss of weight, palpitation, dyspnœa, general illness and rapid pulse after the injections, and where improvement immediately followed a change to 200-300 c.c. at shorter intervals. This intolerance, he thinks, is especially likely to occur where the other lung is of doubtful soundness, or where the digestive organs suffer disturbance owing to descent of the diaphragm. Some patients experience discomfort or even distress for mechanical reasons, if a long interval is left between injections, especially, in the writer's experience, in cases of long standing and with thickened pleura. One of Saugman's cases preferred to continue the treatment indefinitely on account of the discomfort of leaving off.

The rapidity of absorption of gas from the pleural cavity diminishes with time as already noted; it also varies apparently with the amount of lung disease, and certainly with the activity of the patient. It has been commonly stated that the more advanced the disease

the greater the absorption—possibly there is a certain amount of dyspnœa in these cases. The activity or quiescence of the patient exercises a marked influence on the rapidity of absorption of the gas, particularly, perhaps, in cases of partial pneumothorax. In a patient at work, or otherwise “at large,” it is often possible to gauge the amount of his activity by the experiences of the next refill. This supplies an indication for restriction in cases where absorption is inconveniently rapid. But the influence which bears most closely on the question of gas absorption is the presence of pleurisy, whether past or present. At the time of a pleural attack absorption is often nearly in abeyance—indeed the intrapleural pressure may rise apart from the appearance of demonstrable fluid (see Pleurisy). The pleural changes also which follow an attack of inflammation generally lead to a permanent fall of absorption so that in some cases refills may be left to intervals of two months or even longer. One of the writer’s patients who sustained a pleurisy some years ago and is left with a thickened pleura loses now only some 20 to 25 c.c. of N per week. Thus :

5.1.15	.	.	+ 2 + 8	175 c.c.	+ 16 + 21
23.3.15	.	.	+ 1 + 7	200 c.c.	+ 17 + 21

a loss on this occasion of only 200 c.c. in eleven weeks.

As a rule the intervals, gauged by the indications given above, lengthen themselves into a week after the first one or two months, reach two weeks after three or four months, and thereafter expand themselves till three or four weeks can be left between. This is the average interval at which refills need to be done after cases are well advanced in treatment—only after pleuritic attacks can the intervals be carried further than this as a rule.

Intervals in Partial Pneumothorax.—Here, on account of the smaller size of the pneumothorax and the effect of the still mobile lung on absorption, fillings

will generally require to be more frequent than in cases of complete pneumothorax. For a long time fillings at one or two weeks' interval may be needed to maintain the maximum collapse, but after long periods, and particularly if pleurisy supervenes, these intervals may often be lengthened to three or four weeks without disadvantage.

INTRAPLEURAL PRESSURES

Complete Pneumothorax.—In a complete or nearly complete pneumothorax a quite small positive pressure, + 3 to + 5 cm. H_2O , or at most + 10 cm. mean pressure, is generally sufficient to maintain complete collapse. Saugman recommends pressures of + 5 + 10 + 15 cm. according to the individual needs, but considers a mean pressure of + 10 + 15 very high for a complete pneumothorax with normal pleura. He advises that during the first four to six fillings the pressure be kept low, from + $\frac{1}{2}$ to + 2 or 4 cm. mean pressure, and this in some cases will suffice to maintain collapse. But in many cases a higher pressure will be required for this purpose, and very different pressures are needed in different patients even where the pathological conditions appear to be closely similar. The difference appears to depend, even in cases of complete pneumothorax, on conditions of pleural thickening, and it fortunately happens that the need for a high pressure is generally accompanied by the presence of a resistant mediastinum. It cannot be too strongly insisted that the optimum pressure is the *lowest* pressure which will suffice to maintain the lung fully collapsed. There is a curious temptation, which must be recognised and resisted, to raise the intrapleural pressure in artificial pneumothorax above the strictly needful level. This depends largely on the increased tolerance of the patient which will permit a gradual rise to pressures which are needless, undesirable, or even dangerous. For it must be recognised that a

pressure which causes no immediate discomfort to the patient may yet be a source of ultimate danger by displacement and embarrassment of other organs, and particularly of the opposite lung. The danger of attaining a too high pressure exists particularly, the writer thinks, during the first few months of treatment when the pressure-optimum is being sought for—it must always be remembered that it is an optimum and not a maximum that we are out to find, and that at the lowest pressure possible. When this has been discovered, it should be strictly adhered to at subsequent refills. It has been demonstrated that high positive pressures as a rule fall rapidly during the first few hours after a filling; nevertheless the rise to a too high pressure even on a single occasion may, the writer believes, cause a permanent stretching and displacement of the mediastinum, which will remain an embarrassment to the opposite lung even after a return to lower pressures. The effect of such displacements on the mediastinum, diaphragm, and neighbouring organs will be more fully discussed under another heading (see Chap. X.).

Partial Pneumothorax.—In cases of partial pneumothorax much higher pressures can be tolerated and are needed than those suitable for complete pneumothorax. Here the indication is to enlarge the space and to render the pneumothorax complete, or at least efficient. To achieve this result refills should be given at short intervals, and the pressure gradually raised with due attention to the effects on surrounding structures, particularly the mediastinum. In a small gas pocket decided positive pressures may be reached without harm at the first refill, and these may be subsequently increased. Often a gradual stretching or separation of adhesions shows itself in a gradual fall of pressure and the occurrence of marked pleural pain. This may be situated at the base of the chest, or, if diaphragmatic, be referred to the shoulder through the reflex association of the phrenic with the fourth or fifth

cervical nerves (Mackenzie). Occasionally a rapid stripping of adherent pleural surfaces announces itself by a sudden fall of pressure accompanied by pleural pain during the course of a filling.

E. W., a patient with advanced one-sided disease and marked basal cavitation. Pneumothorax initiated by puncture in second space, and 400 c.c. of gas introduced. Refill three days later, when it was found that the breath sounds, before weakened, had returned over the region of puncture. The needle was entered close to the first puncture, a manometer reading of $-12-7$ obtained, and the gas bottle opened. But gas did not flow in, and when pressure was raised the intrapleural pressure rose correspondingly. It was concluded that a small pocket had been entered. The needle was withdrawn and reinserted in the track of the initial puncture of three days earlier. Here a reading of $-5-1$ was found, and on raising the pressure again no gas entered. It was then concluded that the pleural surfaces were in contact and adhesive, and the pressure raised till $+14$ was reached. At this point the patient remarked on a "pricking" sensation, not amounting to pain, experienced in the chest at the site of operation. At the same time gas began to flow in readily, while in spite of this, the pressure steadily fell till after 600 c.c. had entered it stood at $+1+6$.

Pressures of 20 cm. H_2O or thereabouts will often suffice to achieve the desired enlargement of the pneumothorax cavity (see case on p. 55), but with a resistant mediastinum and diaphragm mean pressures of 30 to 40 cm. may be used in some cases, and thereby strong adhesions gradually stretched or separated. Such pressures must not, of course, be reached where they lead to marked displacement of mediastinum or diaphragm, or ballooning of pleura (pp. 104 to 108), but often these parts are stiffened by pleural thickening and offer a good resistance to pressure. In cases where the pleural pocket is in close proximity to the heart high pressures may lead to dyspnœa, pain, cyanosis, and a small, slow pulse on occasion (Brauer, Weiss). The following case of the writer's illustrates this condition, though no distress was observable save some pallor and a sensation of "tightness" at the first filling:

A. A. G., aged 39, with a $3\frac{1}{2}$ years' history, showed moist sounds over the whole right side with a cavity at the apex, but left side practically clear.

On 31.3.14 three attempts to find a free pleural space failed, but on 20.4.14 at the second puncture, free pleura was found in the nipple line, and a small gas pocket produced between this point and the heart. After 150 c.c. of gas had entered marked cardiac pulsations of 3 or 4 cm. amplitude appeared, and obscured the respiratory fluctuation. 200 c.c. were put in and brought the pressure to $+8 + 12$, when the patient became somewhat pale and complained of a sensation of "tightness." The pulse remained 76, the heart was displaced $1\frac{1}{2}$ inches. On three subsequent occasions gas was put in in quantities between 110 and 300 c.c., and the pressure raised to $+38 + 41$ with marked cardiac pulsations without any further discomfort; the gas pocket, however, failed to extend and the attempt was abandoned.

It cannot be denied that a certain amount of danger exists that under high pressures lung-containing adhesions may be torn and embolism result; Saugman also records a case where the patient died after a refill at a pressure of $+40$ cm. with symptoms suggestive of heart failure. The desirability of running such risks—and they may be justified by the patient's otherwise hopeless condition—must be duly considered for each individual case. Sometimes it becomes quickly apparent with a small unyielding pocket that even the highest pressures will achieve no useful result. This has generally been so among the writer's cases where no more than 300–350 c.c. can be put in at each filling (see cases on pp. 53 and 54). It is remarkable, on the other hand, how greatly in certain cases a small pneumothorax can be enlarged with patience even at such pressures as 20 cm. or thereabouts (see case on p. 55).

Effect of Altitude on Gas Pressure.—Since tuberculous patients are particularly partial, with some reason, towards treatment in the high Alps, it is needful to remember the effect of altitude on the gas in a pneumothorax. It has been calculated that a rise of 1,000–1,500 metres (about 3,250–5,000 feet) is equivalent, in a full pneumothorax of 3 or 4 litres, to adding 400–

800 c.c. of gas. This may suffice to cause a dangerous or even fatal intrapleural pressure, and pneumothorax patients must be warned of the dangers of such changes of level. Before going to the mountains their pressure must be suitably adjusted and the patient warned to respect dyspnœa as an indication to break the journey, and abstain for a time from higher elevations.

CHAPTER VII

PHYSICAL SIGNS IN ARTIFICIAL PNEUMOTHORAX

THE physical signs over an artificial pneumothorax often fall far short of that classical picture drawn for the student from cases of spontaneous pneumothorax. After the first filling with some 300 c.c. of gas an area of resonance and weak breath sounds may be discovered to careful examination, and, in addition, an encroachment on the complementary pleural spaces, especially the incisura cardiaca on the left side. These signs increase at subsequent refills, râles and crepitations diminish and disappear, and when the pneumothorax is complete the following signs are present.

The affected side increases somewhat in size, and the intercostal spaces widen even when before treatment these were narrowed. An increase of size of $\frac{1}{2}$ to 1 inch above that of the opposite hemi-thorax may be indicated to the tape-measure. Forlanini describes the appearance of a network of veins over the thorax and upper limb on the affected side, but the writer has not observed this among his cases.

The percussion note varies according to the pressure; if this is low, the side of the chest is hyperresonant, but when the pressure is raised the note becomes high-pitched and even tympanitic. The percussion note over the healthy lung also varies according to the amount of mediastinal displacement. Where this is not much a normal resonant note is maintained, but where a weak mediastinum is carried far over and the healthy lung

relaxed, the note over it will be high-pitched or even, a somewhat ominous sign, tympanitic. It will thus be seen that the comparative resonance of the two sides varies according to the conditions of each, and is often different before and after a refill, and also somewhat for front and back of the chest.

The outline of the pneumothorax can be obtained with fair accuracy by light percussion—auscultatory percussion advocated by Morelli and also by Saugman has not proved of much assistance in the writer's hands.

The breath sounds over a pneumothorax are rarely or never wholly lost, even when the lung is completely collapsed, being conducted in this case from the air tubes or opposite lung. Over the good lung the breath sounds commonly become harsh or puerile, but in some cases they are but little altered from the normal. The distant breath sounds over the pneumothorax side may be of normal quality, but after a time they tend to become amphoric. This change may appear as early as the second filling, but is often delayed for a couple of months or longer. The metallic clang or bell sound, also, is a late sign in these cases, is seldom present in the degree common in spontaneous pneumothorax, and is not infrequently absent altogether. It is always of later appearance than the metallic breath sounds, and is found at first only after refills. According to Saugman it is earliest seen in wasted cases with considerable lung infiltration. A metallic echo of the voice sounds, metallic whisper, and metallic râles or tinkling may also be present.

Râles and crepitations over the diseased lung disappear, as would be expected, when this lung becomes collapsed. Where the pneumothorax is incomplete they may be retained in greater or less degree. It is a remarkable fact, also, that the production of a pneumothorax will often abolish crepitations from the opposite lung, notably from the apex and from the mid-dorsal region, suggesting that some of these signs were in all probability conducted from the diseased side. The conduction of lung

signs, both râles, particularly if consonating, and also bronchial breathing, is a well-known phenomenon in children's chests, but has perhaps received too little recognition in the case of adults. The writer thinks that this explanation applies more to signs over the mid-dorsal region, even out to the axilla in some cases, than to the apex. The disappearance of signs in this latter region he is more inclined to ascribe to the effects of relaxation of the lung due to mediastinal displacement, since they often bear no relationship to signs at the opposite apex. In cases where considerable mediastinal displacement has occurred, crepitations may appear from time to time at the base of the opposite lung, and must be regarded as a danger signal. Such signs are probably, in some cases, atelectatic, but in others almost certainly due to œdema, and further reference is made to them under another heading (p. 112).

Vocal resonance is often remarkably little affected by the production of an artificial pneumothorax; it may remain as strong or nearly so as on the healthy side, but it is commonly somewhat diminished, may be nasal, and, in some cases, as already remarked, is metallic or accompanied by a metallic echo. The vocal vibration, also, is often quite strong on the pneumothorax side, though a rise of intrathoracic pressure may diminish it. If fluid appears the vocal vibration is lost over this, but quite good over the gas above it.

In artificial pneumothorax the collapsed lung can often be mapped out by careful percussion in the inter-scapular region between the first and the seventh rib as it lies along the spine, and it might be supposed that the physical signs, particularly as regards conduction of breath or voice sounds from air tubes or opposite lung, might be different in this part of the chest to elsewhere. This, however, is not the case, and no blowing breath sounds, bronchophony or other signs of condensed lung tissue are present as a rule. Where, however, the lung apex is adherent, harsh or blowing breathing, and perhaps crepitations may be audible here and in the inter-

scapular region, and sometimes also across the spine in cases where mediastinal displacement is a notable feature. These signs may only appear with that pulmonary exacerbation or congestion which so often

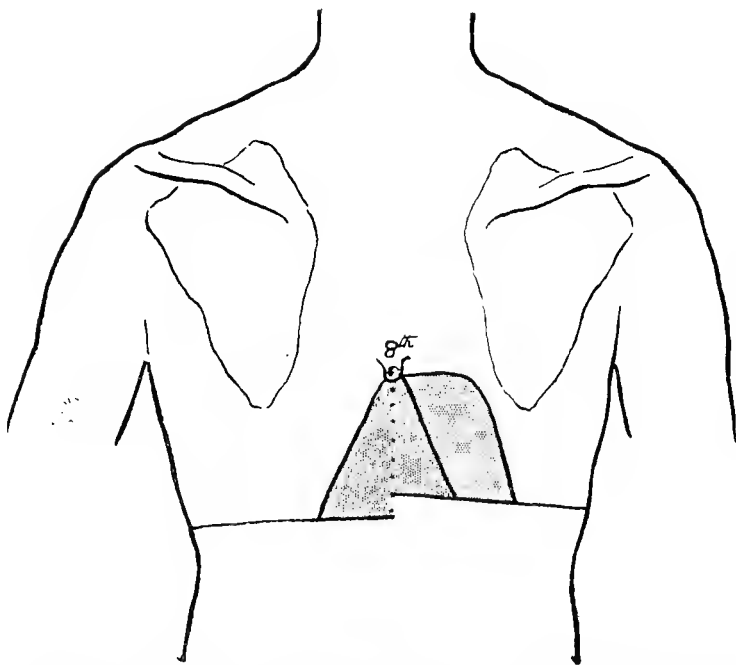


FIG. 5.—SHOWING THE PARAVERTEBRAL TRIANGLES, AND, ON THE SIDE OPPOSITE THE PNEUMOTHORAX, A FURTHER QUADRANGULAR AREA OF IMPAIRMENT HITHERTO UNDESCRIBED.

accompanies the onset of pleurisy, and may be absent or less striking during the remainder of the treatment.

It is of some interest that the basal paravertebral triangle of percussion impairment, associated in disease with the name of Grocco, but also present in the healthy, is still to be found over the pneumothorax side, showing that, in normal chests at least, its explanation must be

found in thoracic muscle and not in pulmonary congestion, as some would have us believe. In addition there is present, on the healthy side, a quadrilateral area of impairment not hitherto described, and not easy of explanation (fig. 5). This has its upper limit at the eighth dorsal spine, the common level of the paravertebral triangle in the healthy, and it overlaps this latter by an inch or two at the base. Its width increases after a refill suggesting some relationship to mediastinal displacement. In cases of pneumothorax with effusion this quadrilateral area of subresonance is greatly increased in size, and reaches up to the fluid level in similar manner to Grocco's triangle in a simple pleural effusion.

CHAPTER VIII

GENERAL RÉGIME: THE COURSE OF TREATMENT AND ITS TERMINATION

DURING the early course of pneumothorax treatment rest in bed is required—this must be maintained at any rate till the pneumothorax is complete (ten days or so), but in many cases is best continued longer. Where a patient's general condition is good, and fever has quite gone, a fortnight's bed may suffice, but even here the displacement of organs makes it wise to proceed slowly from bed to armchair, and so gradually onward. Where prolonged fever and illness have undermined the patient's health a month or more in bed may be needed, and subsequent progress must be even slower and most carefully graduated. A time soon comes, however, in a favourable case, when the patient can be up and about, and then appears one of the most gratifying of the immediate changes achieved by pneumothorax therapy. For whereas before treatment the patient's movements were severely limited by the advent or increase of fever and symptoms on the slightest excess, he can now proceed to lead an ordinary life without any signs of autoinoculation. This enables him quickly to regain his lost physical and mental health, and to bring his muscular system once more into condition. Briefly, he achieves in a few weeks or months the same general improvement which under a spontaneous cure could only be accomplished after years. The whole effect of this is startling and most beneficent, providing the patient is prevented from carrying his energies too far at first. Finally he

can return to a fairly ordinary existence, and will often undertake his original occupation with impunity. The more immediate results of pneumothorax therapy thus divide themselves conveniently into three stages, which may be separately considered under the headings of

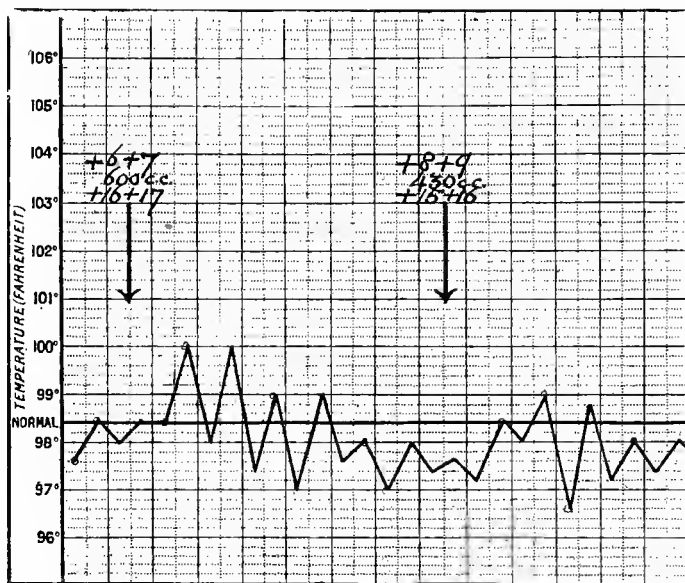


FIG. 6.—ANTOINOCULATION EFFECTS IN A CASE OF PARTIAL PNEUMOTHORAX, SHOWING GENERAL AND ALSO FOCAL REACTIONS.

“Disappearance of Symptoms,” “Clinical Recovery,” and “Anatomical Recovery.”

DISAPPEARANCE OF SYMPTOMS

The immediate effect of the first few fillings is generally an increase of cough and sputum, slight dyspnoea, often with some increased rapidity of respirations, and a sensation of tightness in the chest which may, in some cases, amount to pain where adhesions are present. Since decided pain at a filling is generally an indication

to stop, this should not be a prominent feature. In some cases, also, a noticeable autoinoculation leading to fever, malaise, and other symptoms may follow the fillings before collapse of the lung is complete (fig. 6). These symptoms of autoinoculation are apt to be striking in active cases where the other lung is not innocent of disease, and by leading to increased sensitiveness may start fever which collapse of the worser lung will not suffice to stop (see fig. 7). In such cases

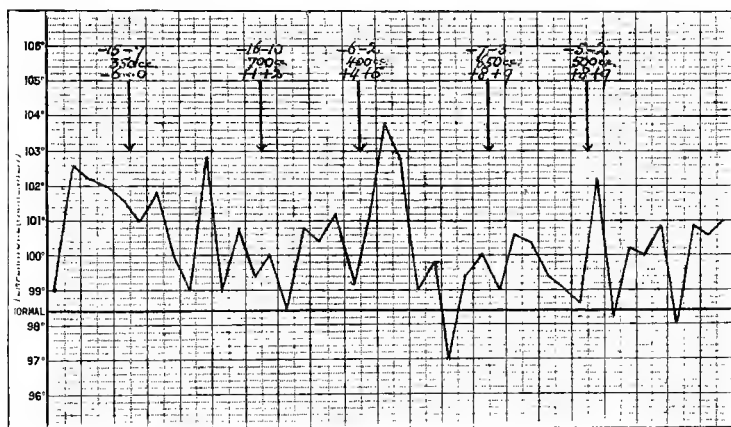


FIG. 7.—AUTOINOCULATION EFFECTS IN A CASE OF BILATERAL DISEASE.

it is necessary to proceed with great care and to avoid refills on the top of an autoinoculation. Where the functioning lung is sound, no importance need be attached to these fluctuations, since the temperature will certainly fall when collapse becomes complete.

Temperature and Pulse.—Soon, in a favourable case, all the symptoms present before the beginning of treatment lessen or vanish, and the patient begins to experience an improvement in many different particulars. Thus loss of fever occurs in cases where the other lung is fairly clear—the drop of temperature,

and closing up of the diurnal fluctuation may be of dramatic suddenness, and occur the day after the second or third filling (fig. 4, p. 58). More commonly it comes about gradually, and may at times be delayed for weeks, even three or four weeks in some cases (fig. 9, p. 91). In some, where other sources of autoinoculation exist the temperature does not respond or only responds gradually or partially. At the same time as the fever, the pulse drops to 70-80, and other

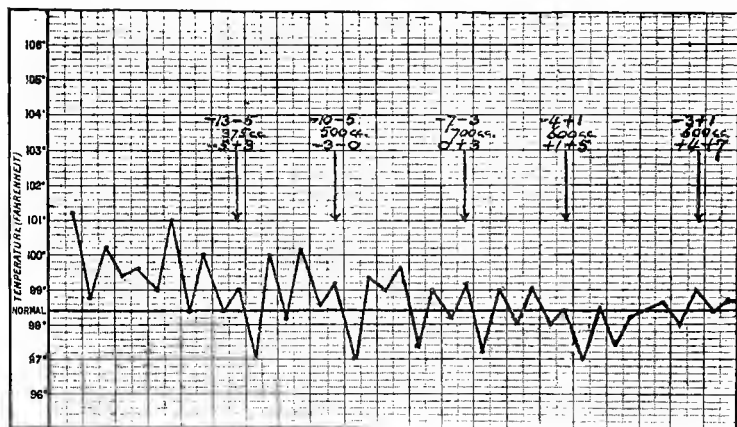


FIG. 8.—CHART SHOWING FALL OF TEMPERATURE IN A CASE OF COMPLETE PNEUMOTHORAX.

toxæmic symptoms diminish, general malaise disappears, sweating ceases, appetite improves, and a general increase of vitality and well-being follows. Dumarest remarks that *dyspnœa* present before the operation often disappears as the result of this treatment, thus showing itself to have been of toxic origin, and with this experience von Muralt and Burnand are in accord. In one of the writer's cases this diminution of respiratory distress was a very notable feature, and was spontaneously remarked on by the patient on repeated occasions.

Cough and Expectoration are increased after the first

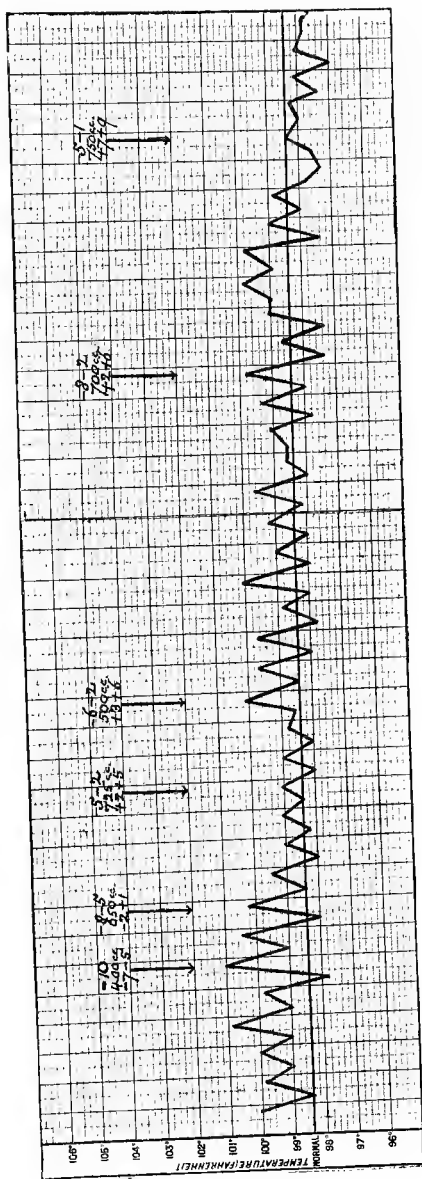


FIG. 9.—CHART SHOWING DELAYED DEFERVESCENCE OWING TO LUNG COLLAPSE REMAINING INCOMPLETE UNTIL A PRESSURE OF $+7 + 9$ CM. H_2O WAS REACHED.

few fillings from the mechanical squeezing out of sputum by the collapsing lung. The expectoration may be doubled or trebled during the twenty-four hours succeeding a refill; but in young patients no sputum may be produced. As soon as the lung is collapsed, the cough and sputum notably diminish or entirely cease. Where collapse is complete the sputum may disappear within a week or two; where some expectoration remains this is either because collapse is incomplete or it may come from central lesions in the functioning lung. As a rule, after diminishing to a fixed quantity there occurs also a change in quality from thick viscid pus to thin, whitish mucus, which floats on the surface of the fluid in the spit cup. Tubercle bacilli diminish and generally disappear in one to six months.

The Weight under pneumothorax therapy is often the least satisfactory item. Young subjects commonly gain well from the first in the writer's experience, but in some cases, and especially in older patients, the weight rises slowly, if at all, and even where a patient regains his health completely the weight may never reach its former level. Sorgo records a loss of weight in 70% of his patients. This depends largely, in the writer's opinion, on the removal of autotuberculin, which, though a fatal poison in excessive quantity, is, nevertheless, in suitable doses, a most potent stimulant to appetite, digestion, and general nervous tone. This possible explanation seems to be upheld by the experience of certain pathologists that there is, after a time, a very notable fall of antibody content in the blood of patients undergoing pneumothorax therapy. During the stage of disappearance of symptoms the patient must be kept under control and gradually initiated into the second stage, which in a favourable case will follow after a longer or shorter interval.

CLINICAL RECOVERY

This is the stage when the patients have so fully lost their symptoms and recovered their general health as to

be capable of returning to a more or less ordinary life. Nothing is more difficult than the decision how far such patients, free of autotoxæmia and consequently in good general health, but yet the subjects of imperfectly healed tuberculous lesions, may be permitted to resume ordinary occupations. In particular it is difficult to decide what risk they run by exposing themselves to the mixed infection inseparable from the neighbourhood of crowded communities. In practice the writer has found the risks of mixed infection enormously less in cases undergoing pneumothorax therapy than in phthisis otherwise treated. Many of his patients are pursuing their ordinary occupations in London, apparently without harm, and when they suffer from a catarrh it seldom seems to give rise to troublesome symptoms. Nevertheless, the writer has convinced himself that these patients, on the whole, do better and are safer living a country life, and most are themselves aware of this fact. Some, free from cough and sputum, find that this returns when they come to town for a few days—it is quite possible that short and occasional visits are of more risk than a prolonged sojourn, when they acquire some immunity to town influences. In cases where a catarrh arises it is very commonly confined to the upper air passages or only descends to the larger tubes, and appears to do no harm. It may be, however, in some cases the starting point of pleurisy, and where an effusion is already present, the writer has, on more than one occasion, seen the fluid and symptoms increase under the influence of a catarrh apparently confined to the upper air passages. It must be remembered that bronchitis is a serious, and pneumonia a fatal complication of these cases, where only one lung is available for the carrying on of vital functions. Nevertheless, Forlanini and Balboni have each recorded a case of successful issue from attacks of serious bronchitis. The rule should be, the writer holds, in cases of pneumothorax therapy in the stage of clinical recovery, that town life should be avoided unless it is clearly necessary, and that

a healthy open-air régime should be followed as closely as possible. In cases where the other lung has been affected or is of doubtful stability prolonged sanatorium treatment or its equivalent may be indicated. Where the other lung is healthy, and the patient has reached a good level of fitness, a return to work is possible or even advisable so long as his working conditions are moderately good in respect of fresh air and exercise. Heavy physical work is contraindicated, though the amount of physical exertion of which some of these cases with a single functioning lung are capable is truly astonishing. Heavy physical work carries with it some risk of perforation of the lung in artificial pneumothorax, particularly in cases of partial pneumothorax, and at the time when a fresh filling is becoming necessary (see Perforation of Lung). A minor disadvantage of marked physical activity, but one which is occasionally a contraindication, is the hastening influence it exerts on the absorption of gas.

ANATOMICAL RECOVERY

This, the last stage in a successful case, arrives at some unknown moment in the course of clinical recovery. Since the collapsed lung is entirely out of reach of investigation it follows that the changes in it can only be, in any individual case, the subject of conjecture. The decision of when to leave off treatment, and allow the lung to re-expand, is the decision of when anatomical recovery, complete cure of the tuberculous process, is judged to have occurred. This decision can, in most cases, only be based on an arbitrary time limit, this limit being arrived at as the result of a compromise, for on one side lies the risk of expanding too soon, and on the other certain disadvantages liable to follow collapse too long maintained. It will be obvious, when so much has been said, that the decision must always be a very momentous and difficult one. For, when the lung is allowed to expand, it may and often will become

firmly adherent to the chest wall, and where active disease is found to be still present, all attempts to obtain a fresh collapse may end in total failure. This risk is one very difficult to avoid since it cannot be decided early, not as a rule before the lung is well expanded, whether disease is still active or likely to become so. To meet this difficulty Saugman recommends that in cases of doubt small injections of gas be continued at intervals pending a definite decision. The expanding lung presents blowing breath sounds, and numerous râles and friction sounds to the stethoscope, even in cases where disease is cured (see p. 99), and it is only by a return of symptoms or the discovery of tubercle bacilli in the sputum commonly present, that the presence of active disease can be definitely proved. By this time the pleural surfaces, if they are going to adhere, will, apart from the precaution suggested above, in all probability be firmly glued together, and efforts to separate them will fail. Adhesion of the pleural surfaces does not, however, occur in all cases. Forlanini declares that cases without complications generally expand without the surfaces becoming adherent. Even where pleurisy with effusion has been present this may sometimes occur. Thus Pigger records a pneumothorax of two years' duration, and complicated by a large exudate, where after some years of health a recrudescence of disease led to the production of a spontaneous pneumothorax in the original pleural space.

On the other side of the decision is the consideration whether, by maintaining collapse too long, expansion, or at least complete expansion, of the lung may not be jeopardised. In this matter there seems little doubt that the time factor, *per se*, can hardly be held responsible for failure of re-expansion; even after short periods, and in the absence of disease, fibrosis may come about. Experiments on animals, by Bruns on rabbits and goats and by Kaufmann on dogs, have shown that even a few months' collapse of healthy lungs may lead to such an increase of connective tissue round vessels,

bronchi, and in the septa and pleura, as to interfere seriously with expansion. The same has been noted among human material. Dunin found in the lungs of cats collapsed two months, and of man collapsed one and a half months, a strong growth of fibrous tissue round vessels and bronchi with complete atelectasis of the alveoli. Burekhardt remarks, in describing a case of artificial pneumothorax of nine months' standing—"the bronchi are likewise grown through and obliterated (in the diseased upper lobe); their smaller branches are at the same time compressed. The vessels are also compressed by fibrous tissue. The same process shows itself in the bronchi of the otherwise sound lower lobe, and might very well have prevented its expansion." In some cases, on the other hand, but little fibrosis occurs. Saugman describes two cases where there was decidedly more fibrosis in a lung five months collapsed, than in another lung collapsed a year and a half (see also pp. 108 to 111).

On the clinical side Forlanini records a case where after eight years' collapse the lung was capable of complete function, and was able to maintain the respiratory needs while a pneumothorax was produced on the opposite side. Evidence from other sources supports the conclusion that complete function may be restored after years of collapse. In many cases, however, it is admitted that expansion has been imperfect after much shorter periods, apart from the presence of extensive disease. Most important in this respect is the influence of pleurisy if this supervenes. Here the time factor is of importance since the longer collapse is maintained, the greater is the likelihood of pleurisy, that common complication of pneumothorax, arising, and it is this which, comes it late or early, constitutes the main bar to complete re-expansion of the lung. For where, with complete collapse, pleurisy has occurred, as it does in nigh 50% of pneumothorax cases, it is more than likely that the lung will be so bound down that full expansion will be unattainable, and this must

needs be a matter of reproach if the disabled lung was the seat of only localised disease. It is for this reason, mainly, that the writer would discourage the use of pneumothorax treatment in all cases of early lung disease.

TERMINATION OF TREATMENT

Having thus outlined the pros and cons which guide us in the decision to suspend treatment, we may consider their practical application and the phenomenon of re-expansion of the lung.

Saugman puts his recommendations as follows :—

A Partial Pneumothorax should be maintained for two years at least, and often much longer, since the less complete the collapse, the smaller the therapeutic effects and the longer its application is required. In some cases treatment may need to be continued indefinitely.

Complete Pneumothorax.—Where disease has been *acute* and of short duration, one year may sometimes suffice, but two years is safer and should be employed if possible.

Where disease is *chronic* two years is the shortest possible limit, and three or four years are safer. A few cases where “the process has been of a more profound or malignant nature” will need to be treated for still longer periods, or even permanently. In the latter case four to six fillings a year will generally suffice to maintain it.

Saugman’s “successful” completed cases recorded in 1913 were actually treated for the following periods :

Under 6 months	.	.	1 case.
$\frac{1}{2}$ -1 year	.	.	4 cases. All acute cases of only a few months' duration.
1-1 $\frac{1}{2}$ years	.	.	5 cases.
1 $\frac{1}{2}$ -2 years	.	.	8 „
2-3 years	.	.	1 case.
3-4 years	.	.	1 „
			20 cases.

Forlanini also recommends an indefinite prolongation of treatment where disease has been extensive and the lung is reduced to a fibroid mass. This will suffice to prevent chest deformity and displacement of organs. In slighter cases he recommends treatment for one or two years according to the severity of disease, but never shorter. V. Muralt recommends at least two to two and a half years of collapse. Even after these long periods disease may break out afresh, while this becomes almost a certainty, if only a short period is permitted. Thus Zink among sixteen cases which got worse attributes this result in five of them to too short a period of collapse ($\frac{1}{2}$ year, $\frac{3}{4}$ year in 2 cases, 1 year in 2 cases), though the first effects of treatment were excellent. Saugman describes two cases of severe disease treated for $3\frac{1}{4}$ and $3\frac{1}{2}$ years respectively, in which disease returned after their re-expansion. Obviously the amount and chronicity of the disease must be the main factors in the decision, since the other factors which influence the healing process in a collapsed lung are entirely outside our knowledge. If the indications for the production of a pneumothorax laid down in this volume are followed, it may safely be asserted that two years should be the minimum length of the cure. Since the patient during the latter periods of the treatment is generally in normal health, and leading a life of reasonable activity, a year more or less where refills are given at long intervals makes little difference to him one way or the other.

RE-EXPANSION OF THE LUNG

When re-expansion is decided on, it is best arranged for the spring months, so that the most favourable time of the year may be utilised for the experiment. Gas must not be withdrawn, save in rare cases where it is urgently required to collapse the opposite lung, but only the refills suspended. Meanwhile cough,

expectoration, and temperature must be carefully followed and frequent examination of the chest carried out. Morelli studies the expansion of the lung experimentally by placing the patient in a positive pressure chamber, and attempts by this means to decide its fitness or otherwise for the proposed cessation of treatment.

The manner of the lung's unfolding will depend on the condition of the visceral pleura and the amount and position of the intrapulmonary fibrosis. The healthy lobes are first freed, and after these, those portions where disease was least advanced. The first clinical evidence of expansion exists in the appearance of faint bronchial breath sounds, and these gradually approach nearer and nearer to normal. Nearly always râles appear, and those of every variety, but mostly small and dry. As soon as the pleural surfaces meet friction sounds, expiratory and inspiratory, and both fine and coarse and sometimes palpable, appear, at first localised, but soon over the whole chest. These friction sounds disappear with time, often remaining three or four months and leaving in some cases a smooth pleura, but in others an adherent lung. This point can be decided with some certainty by examination of the tidal movement of the lung in the lower pleural reflection. In cases where the lung remains cured, the râles also after a time vanish, save where a fixed râle may accompany bronchitic deformity. If they get larger, and especially if they become moister in quality, it is probable that disease is still active, and some gas must be kept in while definite evidence is awaited. In these cases if the lung has to be re-collapsed, no harm appears to follow its temporary re-expansion.

When the lung has reached its full expansion, this will be found to vary according to the amount of disease originally present, the amount of intrapulmonary fibrosis that has occurred—an entirely uncertain factor—and the condition of the visceral pleura. With disease of moderate extent a complete expansion may come

about ; with advanced disease this is hardly to be expected or perhaps desired.

Where the lung will not expand, or will only expand partially, nature demands that the intervening space be somehow filled up. In such cases dislocation of neighbouring organs, the heart and opposite lung, ascent of the diaphragm, and falling in of the chest wall, help to obliterate the pleural space. In many such cases a certain amount of gas remains even after years. To avoid undue deformity and harmful dislocation of organs, Forlanini advises that in such cases refills at long intervals be given permanently, and this is undoubtedly the best course. The patient may himself demand this on account of the discomfort which often accompanies the development of a marked negative pressure in the pleural space (and it may reach -50 cm. H_2O or even more) with its resulting pull on surrounding organs. So much for permanent pulmonary collapse or limited expansion, often, but by no means always, the result of pleural thickening. As a rule it is found, in practice, that lungs or lobes with slight disease expand completely and vesicular breathing returns ; where extensive disease was present broncho-vesicular breath sounds remain, and expansion is likely to be incomplete, so that the heart and mediastinum will suffer some displacement. Among Pisani's eighteen completed cases three expanded fully, and their former slight disease was only just recognisable by some weakening of breath sounds, eight showed some amount of permanent collapse and fibrosis of the lung, and in nine pleural complications left adhesions and pleural thickening, or in some a residue of effusion.

CHAPTER IX

PNEUMOTHORAX AS PALLIATIVE TREATMENT

NOT all cases where pneumothorax treatment comes into question will be found strictly suitable for this method, and the question of temporary improvement and relief of symptoms may have to be taken into consideration. The writer has been most careful to formulate elsewhere (see Indications and Contraindications) clear distinctions between suitable and unsuitable cases, with the hope that patients may be given the chance of this treatment at the right moment if, in any particular case, that moment should ever arise. At the same time he took the opportunity to point out in no doubtful terms (pp. 18 and 19) how easily the treatment of doubtfully suitable cases may discredit a method which, in this country at least, must be said to be still on its trial. It is most important that pneumothorax treatment should not be put forward as a panacea for pulmonary tuberculosis of every shade and degree; still more that it should not be considered a last refuge of the dying. Nevertheless it must be admitted that benefit and marked increase of comfort may be achieved in cases which lie outside the limits of strict suitability, and at the risk of a charge of inconsistency, the writer will now devote a little space to the consideration of pneumothorax as palliative treatment.

It will have been noted under Results of Treatment (p. 167) that Saugman recorded great temporary improvement among many of his "quite hopeless" cases, and that two of them even appeared to qualify for a possible recovery. In addition Pisani noted a similar marked improvement among his cases of double-sided disease,

though a spread in the opposite lung followed at a later stage. Forlanini has always been opposed to too rigid an interpretation of pneumothorax "indications": he has himself seen recovery in cases where the opposite was to be expected, and he holds that double cases where there is "everything to gain and nothing to lose" should be given the chance. "When the lesion of the other lung is early and limited, cure is still possible; if it is more advanced one can at least expect a long arrest. Moreover improvement, which in exceptional cases may be truly astounding, is still possible when the lesion is well advanced; in any case one can always count on a disintoxication of the organism—a disintoxication partial and fleeting if you will, but one which, in a patient at the end of his resources, is always a gain."

The writer has treated a certain number of advanced and apparently hopeless cases by the production of an artificial pneumothorax, and can confirm the occurrence of temporary improvement in many of them. In one, a case of rather acute disease, œdema of the functioning lung occurred, and the inevitable end was rather hastened than delayed. But as a rule a reduction of autoinoculation has been achieved without any very serious effects on the opposite lung. The patient has felt better and the temperature has been reduced, though not brought down to normal; in many cases cough and sputum have been, for the time, very greatly diminished. In some cases a pleurisy has occurred at a quite early stage of the proceedings, but this has had the advantage of limiting the absorption of gas and delaying the need for its replacement.

In treating these cases the usual aim of artificial pneumothorax—namely, complete collapse of the lung—must be very considerably modified. The aim must here be to damp down autoinoculation from the most diseased lung without putting an excessive functional and mechanical strain on the other. In cases where fibrosis has displaced the heart and mediastinum towards the most diseased side, a considerable amount

of gas may be put in without more effect on the opposite lung than that of returning it to its original size. This may benefit rather than embarrass—indeed a certain amount of relaxation of the functioning lung over and above this should be actually beneficial, in theory, on account of the hyperæmia which is said to result. In any case the pneumothorax must be enlarged slowly and cautiously, the functioning lung being carefully watched the while. If any increase of signs appear in it this is clear indication for a lowering of pressure in the pneumothorax. By a slow and careful régime an optimum pressure may soon be discovered based on the tolerance of this diseased functioning lung. In some cases the amount of gas tolerated may only be enough to abolish crepitations, while leaving quite audible breath sounds—but with general improvement nevertheless. The aim must then be to keep the pneumothorax as near this point as possible, without too great disturbance of the patient by the need for frequent refills. Fortunately in these advanced cases the gas has been in the writer's experience but slowly absorbed, and a small filling every two weeks or so will generally suffice, particularly if there is a little pleural exudate present. It is especially in cases of this character that marked autoinoculations are apt to follow the refills (fig. 7, p. 89), particularly if the lung can be but partially collapsed. A not uncommon effect of the fillings is to force the patients to lie on the most diseased side, when before they were prevented from doing so by pain. The effect of this is to limit that flow of sputum under gravity out of the lung into the larger tubes which was the cause of cough, and thus to aid the partial collapse in its effects on cough and general comfort.

The treatment of advanced and double disease by simultaneous partial collapse of both lungs appears to have given good results in the hands of certain operators (see p. 18), but the writer has had no practical experience of this method hitherto.

CHAPTER X

EFFECTS OF PNEUMOTHORAX ON THE THORACIC ORGANS

THE MEDIASTINUM

IN man and in certain animals the two halves of the thorax are partitioned off from one another by so strong a mediastinum that even a fully open pneumothorax can be for a time tolerated. On the other hand, in small dogs, according to Bruns, and also in rabbits and guinea-pigs in the experience of Breccia, an open pneumothorax cannot be maintained owing to weakness of the mediastinum and the ease with which gas passes through it.

In an **Open Pneumothorax** the pressure is the atmospheric during both inspiration and expiration. During inspiration the mediastinum, already displaced by the difference of pressure, moves still further towards the healthy side, to return again during expiration when the pressure in the healthy side rises once more towards the atmospheric. At the same time some air may be forced into the collapsed lung during expiration, the pressure in the bronchi having risen above the atmospheric (+ 2 to 3 mm. Hg. or + 2·7 to 4 cm. H₂O), to be withdrawn again during inspiration. As a result of the flapping to and fro of the mediastinum with every respiratory change, the functioning lung can neither expand adequately during inspiration nor completely empty itself in expiration, and the mechanical disabilities brought about by an open pneumothorax soon manifest themselves in dyspnoea and signs of deficient oxidation. If, however, the external opening is smaller

than the calibre of the trachea, the conditions approach more nearly to those of a closed pneumothorax, the lung does not completely collapse and expands somewhat with inspiration, though the movements of the mediastinum may still be of a kind to embarrass the functions of the healthy lung.

In a **Closed Pneumothorax** under pressure the elastic mediastinum is stretched and steadied by its displacement and by the descent of the diaphragm; it thus forms a more efficient partition, and allows the functioning lung to fill and empty itself more easily. Where the pressure in the cavity is high, little or no respiratory movement takes place. When the pressure approaches to or falls below the atmospheric, the mediastinum, contrary to the condition in open pneumothorax, is drawn over to the pneumothorax side during inspiration, owing to expansion of the chest wall rarefying the gas, and returns again during expiration, thus helping the expansion of the functioning lung in place of hindering it. Hence in closed pneumothorax there need be but little or no interference with respiratory comfort, and this is the common experience in practice.

With the formation of an artificial pneumothorax the mediastinum moves over, as already noted, towards the healthy side. Its elastic resistance must balance the difference between the low pressure on the healthy side and the pressure, generally above the atmospheric, at which the pneumothorax is maintained. A bow-shaped mediastinum thus results, generally with the maximum bulging in its lower part, but occasionally exhibiting a weakness above between the clavicle and the third or fourth rib. Three left-sided cases of Forlanini's showed a bulge in this region extending out even to the right nipple line. A case of the writer's, also left-sided, shows a similar weakness in this region.

The resistance of the mediastinum to pressure is a very variable quantity, and is, not infrequently, the factor which determines success or failure in artificial pneumothorax. In some cases pleural thickening supports

the mediastinum so adequately that considerable pressure can be used without displacement occurring, as was the case with the patient whose temperature chart appears as fig. 9, p. 91. In others the mediastinum may be so weak and mobile that its displacement prevents the utilisation of an adequate intrapleural pressure. Over-displacement may occur at or even below atmospheric pressure in some patients. Particularly troublesome are cases where weak areas in the mediastinum give rise to troublesome local bulgings or herniæ. Nitsch describes two weak spots as likely to give rise to trouble:

(1) In the anterior mediastinum at the level between the second and fourth rib articulations.

(2) In the lower part of the posterior mediastinum between the vertebræ and aorta behind, and the œsophagus and heart in front.

At these two points the two pleuræ meet or cross, and for want of support a hernia is not unlikely to form under pressure.

Such local pockets or herniæ may invade the healthy side, and in cases with effusion may even, according to Mayer, simulate a double pleurisy. They must always be a cause of anxiety, and are an indication for the limitation of gas pressure since a rupture might occur, though in practice this seems to be a rare and not very serious event.

In practice it should be insisted that the mediastinum must never be displaced as far as the mammary line, however low the pressure which leads to its dislocation. The amount of its displacement must be kept constantly in sight during the production of full collapse of the lung, since an overstretching is not easily retrieved. Further remarks on this point will be found under the heading of the "Functioning Lung" (p. 113).

THE DIAPHRAGM

The diaphragm under the influence of artificial pneumothorax presents interesting peculiarities deter-

mined to a large extent by the condition of the intrathoracic pressure. When the pressure is high the diaphragm on the side of the pneumothorax is depressed or even inverted, and practically stationary. With a pressure not far removed from the atmospheric, diaphragmatic movement is observed, and this presents a varying picture according to the influence of several factors whose value is as yet but partially understood. Thus diaphragmatic movement may follow that of the healthy side; on the other hand, a paradoxical or inverted diaphragmatic movement may be present. This was first described by Kienbock in cases of spontaneous hydropneumothorax, the fluid being seen to rise in inspiration and fall during expiration. It is found in a proportion of cases of artificial pneumothorax, but only, as was pointed out by Rist and Maingot, where the intrathoracic pressure is above the atmospheric. When the pressure falls below this point the movement ceases, or a normal movement takes its place. In some cases the diaphragm rises at the beginning of inspiration and then moves the other way—a sign that inspiration has transformed a pressure just positive into a negative pressure. Saugman and Hansen have described Kienbock's phenomenon as an early sign of effusion—it is then best studied in left-sided pneumothorax owing to the presence of the liver on the right side. The factors which may determine the production of inverted diaphragmatic movement may be stated as follows:

(1) Inspiratory rarefaction of gas in the pneumothorax cavity tending to draw up a flaccid diaphragm just as it pulls over the mediastinum.

(2) Increased intra-abdominal pressure during inspiration owing to descent of the diaphragm on the sound side tending to raise a flaccid diaphragm on the diseased side.

(3) Contraction of an inverted diaphragm leading to an inverted movement.

Exactly how these factors work, and to what extent

any of them are responsible, and what other influences, if any, may also be at work, is at present but little understood. However it is brought about, Kienbock's phenomenon may be put to practical use as evidence of the height of intrapleural pressure. Its presence in company with an inspiratory movement of the mediastinum towards the pneumothorax side is evidence that the pressure is falling towards the atmospheric. Its disappearance after it has been present shows that the pressure has now fallen below the atmospheric.

THE COMPRESSED LUNG

The compressed lung, when free to collapse, shrinks to a mass the size of a couple of fists or smaller lying against the mediastinum. Its substance is airless and fleshy, "pneumonic foci and old isolated caseous tubercles show a remarkable cicatricial transformation and are surrounded by fibrous tissue. The liquid component of the caseous masses is pressed out by the elastic retraction of the lung and evacuated through the bronchi; the rest is compressed more and more by the connective proliferation, and finally completely isolated in a coat of compact lamellous conjunctive tissue. The caseous substance dries up and often gets calcified. Cavities, if they are situated in a portion where compression has been more or less completely realised, can be entirely obliterated, their opposing walls having been brought in contact and having adhered to each other." Thus Rist, who points out that "a marked tendency to over-production of fibrous tissue in all tuberculous lesions of the atelectatic lung is the characteristic fact emphasised by every author."

But this fibrotic tendency in the collapsed lung does not appear to be entirely dependent on the presence of disease. Kaufmann in the healthy lungs of large dogs found, after $7\frac{1}{2}$ months' collapse, a thickening of the alveolar walls, and inflammatory infiltration and increase of perivascular and peribronchial connective

tissue sufficient, in his opinion, to prevent re-expansion of the lung. Bruns found similar changes in the collapsed lungs of rabbits and goats, and evidence exists that the same process, due perhaps to lymph stasis, may occur also in the human. Thus Burckhardt, writing of a case of artificial pneumothorax of nine months' duration, after describing the obliteration by the growth of fibrous tissue of bronchi and vessels in the diseased upper lobe, remarks: "The same process shows itself in the bronchi of the otherwise sound lower lobe and might very well have prevented its expansion." As a clinical phenomenon fibrosis of collapsed lung tissue, particularly in children, is a quite familiar and well-recognised phenomenon.

Where the lung is diseased, fibrosis is still more a feature of collapse, and forms, so far as local recovery is concerned, the main object of compression in artificial pneumothorax. Graetz was particularly struck with the "unusually marked tendency of the tuberculous tissue towards fibrosis. Alike in the neighbourhood of vessels and bronchi," he continues, "there has arisen a widespread development of connective-tissue hyperplasia which must be regarded as the outcome of healed perivascular and peribronchial inflammation; in addition the pneumonic foci, and in particular old caseous, solitary tubercles, show a marked fibrous change and encapsulation in contrast to the surrounding tissue." He regards the process as directly dependent on the lung compression "particularly since the tuberculous foci in the functioning lung do not exhibit a similar tendency to fibrosis."

But the degree of this change in the collapsed lung also varies greatly in different cases, and this appears to depend more on the completeness of the collapse, and the amount of compression, than on the duration of treatment. On this account Graetz noted more fibrosis in a lung compressed for but 53 days than in another subjected to $6\frac{1}{2}$ months' treatment. He found the fibrous hyperplasia greatest where the intrapleural

pressure came most into play—in small isolated portions of lung substance, at the apex, and in the superficial parts of the large lobes, which exhibited fibrosis to a notably greater degree than did their central tissues. Portions of lung protected from pressure by adhesions, as in partial pneumothorax, showed but little fibrosis. Pneumonic areas, also, were found, for mechanical reasons, less amenable to these reparative changes than were scattered or isolated tubercles. Bruns found the fibrous change to start at the pleural surface, the result, he thought, of irritation by the nitrogen gas, and to dip into the lung in the form of wide septa. Cantani and Arena recorded similar experiences in their experiments on animals. Bruns observed inflammatory infiltration in the neighbourhood of bronchi spreading out round the neighbouring vessels, and this he regards as in part pleuragenous and in part due to infection arising from obstructed bronchial secretion. He considers the process in all cases of inflammatory origin and not due to stasis and irritation of toxins, as Brauer and also Graetz suggest. At the fullest development of the scarring process the lung may be practically converted into a fibrous mass, bronchi and vessels being grown through, compressed, and obliterated. In a case of Forlanini's which died of pneumonia two years after the termination of treatment, "the lung was changed into dense fibrous tissue which enclosed a completely encapsuled focus of old caseous material."

Such a result must be a very undesirable one save in cases where the lung was the seat of advanced and widespread disease, in which circumstance it might be the best that could be hoped for. But in spite of the tendency to fibrosis exhibited by collapsed lung tissue even apart from the presence of disease, it fortunately happens in practice that the collapsed lung of artificial pneumothorax is commonly capable of complete, or at least of useful, re-expansion. As Lillingston has remarked, the lung may fully re-expand without difficulty after many years, or, after only a few months' com-

pression, become a mass of scar tissue enclosed in a fibrous capsule. In practice it may be noted that a good many completed cases are now going about the world with a functioning lung, and in some of these the lung has even been made to give proof of its physiological capacity during the production of an artificial pneumothorax on the opposite side (see also pp. 95, 96, and 100).

THE FUNCTIONING LUNG

During the course of an artificial pneumothorax the healthy or healthier lung must support the whole burden of the respiratory function. This should be no difficult matter if the lung is sound and unhampered, since it was shown by Riva Rocci and Cavallero, and also by Carpi working in Forlanini's clinic, that one-third of the pulmonary surface is sufficient to support life comfortably, at least during rest. Indeed, Bernard, Le Play, and Mantoux found even one-sixth of the lung volume might suffice for respiratory needs. But the functioning lung in artificial pneumothorax cases is commonly the seat of some amount of disease, albeit of a quiet nature, and in addition it is of necessity somewhat hampered, its volume being decidedly reduced by the invariable displacement of the mediastinum. On account of this fact it cannot increase in size (unless to a minimal extent into the complemental pleural spaces) to support the increased work, as some authors have curiously insisted; an increased demand can only be met by an increased rapidity or depth of respiration, and by a fuller circulation. As a matter of fact, the respirations are seldom increased except immediately after an operation, and dyspnoea may be relieved but is seldom aggravated by a well-managed artificial pneumothorax; from which it may be argued that the functioning lung, in spite of its disabilities, is generally well able to maintain the respiratory requirements. As to its blood supply this is probably more ample than

normal, though the evidence on this point is at present of a somewhat conflicting nature (see p. 116).

As already remarked (see Physical Signs), it is no uncommon experience that crepitations present before treatment in the healthy lung disappear or are reduced immediately on the production of a pneumothorax. These sounds have doubtless often been conducted from the diseased lung and disappear with its collapse, but the writer has suggested that in some cases the disappearance of signs may be attributable rather to relaxation of the functioning lung brought about by displacement of the mediastinum.

It is this pulmonary relaxation which appears to be the main threat hanging over cases of artificial pneumothorax. If the mediastinal displacement is excessive, expansion of the good lung is reduced, and symptoms and signs referable to this may arise. In the first place there is evidence that quiet tuberculosis is likely to become active under these circumstances, improvement or arrest being again secured, in many cases, by a reduction of the excessive pressure and dislocation. The more common threat, in the writer's experience, is that of pulmonary œdema, a condition which must be looked for in cases with a yielding mediastinum. In these cases dyspnœa is present on but slight exertion, and the percussion note is high-pitched over the functioning lung. Crepitations appear at the base of this lung and tend to change their position from day to day and with the occurrence of refills. These crepitations may be in part atelectatic and dependent on the difficulty of expansion of the hampered lung, but they are certainly in many cases due to œdema, and in this case are moist in quality and accompanied by cough. Such signs are evidence that the healthy function of the lung is threatened, and are an indication for the reduction of intra-pleural pressure. They will in many cases clear up after weeks or months without such change of treatment, but it is hardly safe to rely on this. In a case of advanced caseous tuberculosis under

the writer's care œdema supervened rapidly and led to death. Here the lung was disabled by active central disease; displacement of the mediastinum was but slight and at most a contributory factor.

It is particularly important that the mediastinum and functioning lung be carefully watched during the initiating of a pneumothorax. Only just such a pressure should be attained as suffices to collapse the lung—a needless rise of pressure on only a single occasion may overstretch the mediastinum and threaten the success of subsequent treatment. For though it is easy to displace the mediastinum by pressure, reduction of such pressure will not necessarily retrieve the situation and bring back the mediastinum. To do this a pressure below that needed to maintain pulmonary collapse may be required. In cases with flaccid mediastinum some skill may be required in making the compromise between a useful intrathoracic pressure and avoidance of over-displacement. The writer has experienced most trouble in cases of left-sided pneumothorax; indeed he has seen signs of pulmonary œdema only on the right side and that altogether in four cases. Two of these were cases of advanced and well-nigh hopeless disease—one has already been referred to as dying of pulmonary œdema. In the other two the right border of the heart extended out to the right nipple line (about 4 inches to the right of the mid-sternal line), and the writer thinks this the limit of safe displacement to the right. To the left the writer has often carried the heart's border out 1 to $1\frac{1}{2}$ inches outside the left nipple line, and even to the anterior axillary line, without any inconvenience arising. Forlanini mentions displacement carrying the heart to the anterior axillary line on the right side or the mid-axilla on the left without any reference to pulmonary embarrassment. A high-pitched percussion note is the best sign of pulmonary relaxation due to encroachment of organs—a tympanitic percussion note is an extreme degree of this and one which should never be experienced in practice.

THE LUNG CIRCULATION

The effect of artificial pneumothorax on the comparative blood content of the two lungs is a vexed question which has found, up to the present, no certain solution. In spite of a common belief that blood is sucked into the lungs during inspiration, it was shown by Poiseuille, as early as the year 1855, that the blood capillaries are, in fact, narrowed during inspiration, and this discovery has been confirmed by numerous investigators of recent years. Cloetta, as the result of careful experiments, plethysmographic, circulatory, and microscopie, has arrived at the conclusion that the further the lung passes from full extension towards collapse, the greater becomes its blood capacity, and this whether the changes of volume are brought about by pressure from within or by suction from without. This would seem to support the view that in cases of artificial pneumothorax it is the collapsed lung which contains the most blood, and Cloetta actually seeks to attribute its healing to the action of such hyperæmia. Results directly opposite to these have been obtained by other observers. Douglas Powell in 1869 conducted circulatory experiments on excised lungs which appeared to confirm the earlier observations of Goodwyn (1788) and Erichsen (1845) in this country, that mere collapse of the lung brought about by its own elasticity does not materially affect the circulation through it. He found, however, that under more complete collapse there was a decided increase in circulatory resistance, and this independently of external pressure (by compressed air). This result, be it noted, does not exclude a passive hyperæmia.

Bruns, working with lungs still in the pleural cavity, and confirming earlier work by Lohmann and Müller on similar lines to Powell's, finds that if the lung is extended by outside suction (as in Sauerbruch's negative pressure chamber) its blood content increases above the normal; the collapsed lung, however, kept under pres-

sure he finds to contain very little blood, the capillaries being mostly empty to histological examination. This view has been supported by Brauer and his followers, and also by Forlanini; these clinicians are of opinion that it is the functioning lung which is hyperæmic, and they look to this hyperæmia as an explanation of the favourable effect which pneumothorax therapy commonly exerts upon the tuberculous areas within it. The collapsed lung is, in their opinion, comparatively empty of blood.

These conflicting views, each supported by experiment, and backed by distinguished authority, seem at first sight irreconcilable. If it is borne in mind, however, how many different stages are passed through between extreme collapse and complete extension, and how each must carry with it its own particular blood state, it seems quite probable that both views represent something of the truth under different physical conditions. It might well be expected that a lung collapsed under high pressure would be comparatively bloodless, and this, as a matter of fact, is readily admitted by Cloetta. He found the lung most hyperæmic when sucked out a trifle from the collapsed state under a negative pressure of -2 to -3 mm. Hg., but admits that a positive pressure might render it comparatively bloodless. Nevertheless, it may fairly be questioned whether it ever reaches this state under the ordinary conditions of an artificial pneumothorax. For Breccia declares that the lung capillaries maintain a pressure two-fifths of the systemic capillary pressure of 40 mm. Hg., and hence can only be affected by pressures as high as 16 mm. Hg., or nearly 22 cm. H_2O , a pressure but seldom reached in cases of complete pneumothorax. Lower pressures than this, he argues, will only obstruct the pulmonary veins, and lead to venous congestion. If these statements are correct it must be concluded that any real ischæmia of the collapsed lung must be of but rare and temporary occurrence; a passive hyperæmia seems the more likely resultant.

In the functioning lung changes of blood content may be expected according to the amount passing through the collapsed lung at any given time, though in this connection it must be noted that both organs may be simultaneously hyperæmic, as, indeed, Breccia has experimentally demonstrated.

For the collapsed lung is very notably diminished in volume, and in addition a passive hyperæmia may be associated with a greatly restricted blood flow. The blood supply will also vary with the amount of extension of the functioning lung. For it must be insisted that the functioning lung is not unduly extended in artificial pneumothorax as some have asserted, but is actually more relaxed than normally, owing to displacement of the mediastinum. Under this influence it seems highly probable that it is under most circumstances, and particularly after a refill, really hyperæmic as held by Brauer and Forlanini, and this view seems to find support from X-ray examination. von Muralt has stated that hyperæmia of the functioning lung is always visible to the rays, and Saugman expresses the same opinion, but remarks that the early shadowing disappears at a later stage, owing to the development, according to his view, of pulmonary emphysema. How far the occurrence of œdema of the lung under excessive dislocation of the mediastinum may be held as evidence of hyperæmia, particularly of venous hyperæmia, must be at present open to doubt. It seems to the writer at any rate a somewhat significant complication.

HEART AND CIRCULATION

The heart in artificial pneumothorax must often submit to considerable dislocation, but it seldom or never gives evidence of functional embarrassment. In the writer's cases with left-sided pneumothorax, the heart's border has commonly reached 3 to 4 inches to the right of the mid-sternal line, at most to the nipple line; in

right-sided pneumothorax some 1 to $1\frac{1}{2}$ inches outside the left nipple line. In other words the organ has moved its position by some 2 or $2\frac{1}{2}$ inches, and this without any symptom of discomfort. Forlanini describes displacement to the right anterior axillary line and to the left mid-axilla respectively without embarrassment, and Saugman describes a case where the apex was palpable in the left anterior axillary line before the operation, and a week later pulsation could be felt in the right nipple line without the patient experiencing the least discomfort. Indeed it seems to be always the tolerance of the functioning lung, and not of the heart, which rules the situation in mediastinal displacement.

Murmurs attributable to cardiac displacement seem, on the whole, rare, and the literature contains but few references to such. Weiss has recorded an aortic diastolic murmur and Carlström a basal systolic murmur. Forlanini has heard both mitral and pulmonary murmurs attributable to deformity, and has found these disappear completely at the termination of the cure. The writer has had no abnormal murmurs appear among his cases. The effect on the pulse frequency is various and conflicting. In many cases an increased frequency follows the gas injections, but Weiss has often experienced a slowing, in one case from 80–88 down to 50–60 due, he thinks, to vagus irritation. The fall of the pulse resulting from the removal of toxæmia is liable to confuse or to counteract any changes depending on circulatory disturbances.

In addition to displacement the heart may be called upon to compensate for a reduction of passage-way through the pulmonary channels, a matter on which some diversity of evidence and opinion at present exists. Bruns in numerous experiments on dogs, goats, and rabbits found hypertrophy of the right heart always followed pneumothorax of three months' standing. Postmortem experience in man gives somewhat conflicting evidence. Right-sided hypertrophy, or dilatation, or both have been found by Carlström, Waller,

Hymans and a few others, but five autopsies by Brauer and Spengler, three by Graetz, and others by Saugman and Hansen and by Kistler revealed no cardiac changes. These discrepancies probably permit a similar explanation to that already offered for the various views of pulmonary blood content advanced by different authorities (pp. 114 to 116). It may well be that in some cases the total pulmonary circulation is in no way embarrassed, while in others a notable reduction of the pulmonary blood channels may be present, and place an increased strain on the right heart, all according to the degree of pressure on the collapsed lung, and the degree of expansion, determined by mediastinal displacement, of the functioning organ. This view seems to receive support from discrepant blood-pressure observations obtained by different observers—Bursten finds a rise of general blood pressure for a short time after a refill, whereas Guichard records a fall of systolic blood pressure of about 20 mm. Hg. with a subsequent rise to normal after twenty-four hours. Kessel and Taschman found no constant blood-pressure changes before, during, or after the gas fillings. Be it noted, however, that the total circulation may be reduced, and yet the functioning lung be decidedly hyperæmic, so that there is nothing contradictory in the discovery by Bruns of hyperæmic lung, but at the same time obstructed circulation, as a result of his experiments on animals.

So far as cardiac enlargement is concerned the writer has long been struck with the width of the deep cardiac dulness in his pneumothorax cases, the transverse measurement being often $5\frac{1}{2}$ inches in place of the $4\frac{1}{2}$ commonly found in health. This he has been inclined to attribute to a dislocation of the heart forwards against the chest wall rather than to true enlargement, since a forward displacement of the heart was noted by Sallé in cases of pleural effusion, and the same might well be expected in pneumothorax. The matter at present remains in some doubt, and further work is needed for its satisfactory elucidation. One of the writer's cases

in whom a good deal of emphysema was present, and in whom the cardiac dulness measured some 7 inches across throughout treatment, died rather suddenly of heart failure after pneumothorax had been kept up for more than a year with otherwise satisfactory results.

CHAPTER XI

PLEURISY AS A COMPLICATION

PLEURISY is so common and so important a complication of artificial pneumothorax that no apology is offered for the amount of space accorded to its discussion in this small volume.

PREVALENCE

It is to be expected in some 50% of cases followed through from start to finish. It is not always an early complication, and it may thus be missed in cases treated for a short time or drifting into other hands. Among ninety-nine effusions recorded by Saugman (in ninety-six patients of whom three were treated on both sides at different times) the fluid appeared at the following intervals of time :

In 21%	within the first 3 months.
„ 33%	„ „ „ 6 „
„ 41%	„ „ „ 9 „
„ 44%	„ 1 year.
„ 50%	„ 3½ years.

It is thus seen that the chance of effusion increases greatly with the length of treatment. von Muralt was also able to show that the character of the case greatly influenced the occurrence of effusion, this occurring more frequently and also earlier among advanced and acute cases. Weinstein of Davos experienced effusions among 70% of advanced cases, among 33% of moderately

advanced, and only 10% of early and slight cases. These factors, no doubt, are largely responsible for the discrepancy between the figures of various observers. Thus the writer experienced no case of effusion within the first eight months of commencing to use this form of treatment—subsequent events have caused him to abandon the hope that this immunity bore any relationship to his methods of operating. Brauer and Spengler had 44% of cases within fifteen months, von Muralt 50% of fresh effusions among 64 cases of long duration, Begtrup-Hansen 50% among 66 cases, Ziegler of Heidehaus about 50% among 60 cases, Weinstein 33% among 71 cases, Tecon and Sillig but 29·63% among 108 cases. Brauns of Hanover mentions but 5 effusions among 98 pneumothorax patients, 80 of whom were cases of phthisis. Mayer punctured and studied 18 exudates among 46 cases, and saw also 4 more too small and fleeting for inclusion among his cases, a total of nearly 48%. It must be admitted that small and fleeting effusions may sometimes be missed, and that particularly where X-ray examination is not to hand; this perhaps may furnish additional explanation of personal discrepancies. In partial pneumothorax pleurisy is much less prone to develop than in those which are complete or nearly so.

CAUSATION

Apart from a few cases of infection with pyogenic organisms, the effusions of artificial pneumothorax are sterile to culture and, for the most part, are found to contain tubercle bacilli either to microscopic examination or to animal inoculation. Saugman was able to demonstrate the tubercle bacillus in all his large and chronic exudates with only two exceptions. When the mechanical conditions present in an artificial pneumothorax are borne in mind, it is hardly surprising that tuberculous pleurisy should be of common occurrence. For whereas with an expanded lung the activities of

surface foci are readily limited by the formation of adhesions, when the diseased lung is collapsed and the pleural layers separated, any small focus of tubercle at the surface, its barriers removed, may run riot and lead to the production of a serous or sero-purulent effusion. This explanation probably covers the main part of the ground, but other influences are at work also which modify and thus tend to specialise the exudates arising in these cases. Thus the effect of separating these normally opposed moist surfaces must be borne in mind, and, in addition, the influence on them of the constant presence of nitrogen gas, an influence which Kaufmann found to lead to histological changes of the serosa in experiments on dogs. The changed gas absorption from the pleural surface during the course of artificial pneumothorax cases is evidence of this influence, and both Mayer and von Muralt tend to regard it as of some importance in the occurrence of pleurisy. The irritating effect of these influences on the pleural surfaces, even after a minimal interval, is well illustrated by the case of E.W. described on p. 79, though it must be admitted that the action of the local anæsthetic, novocaine, cannot be excluded. Here, unadherent surfaces were rendered adhesive after less than three days of separation by a layer of nitrogen gas, and on coming together needed a pressure of $+ 14$ cm. H_2O to strip them apart again. In addition it has been observed by Forlanini, and von Muralt is in agreement also, that often a severe exposure or chilling, or some over-exertion, has preceded the onset of these pleurisies, and has constituted at least an important predisposing cause. Indeed, von Muralt even speaks of pleurisy as an epidemic occurrence among these patients during certain cold months of the year, particularly March and April. The writer has found a certain connection between catarrh of the upper air passages and the onset or exacerbation of pleurisy among his cases. He has also been much struck with the relationship between the more severe of these pleural attacks and evidence of

congestion or of renewed activity in the pulmonary tuberculous foci, and this has been remarked on by Mayer. Pleurisy may also arise from the breaking down of adhesions, and possibly from puncture of adhesions containing a core of lung tissue. Infection of the pleura from without should never occur under proper aseptic precautions ; even the carrying in by the needle of a punched-out portion of skin seldom or never seems responsible for pleurisy—nearly always the piece of skin remains within the lumen of the needle.

DRY PLEURISY

The occurrence of dry pleurisy is an undoubted fact ; it may or may not lead on to effusion. It may be ushered in by pain in the side, vomiting and fever, as in a case of the writer's ; areas of impairment may be found and of shadowing to X-rays, but yet no fluid follow. Where, in these cases, the pneumothorax is only partial, pleural friction may appear and confirm the diagnosis ; or friction may be heard at the diaphragmatic reflection in complete pneumothorax. Among forty cases of pleurisy recorded by L. Spengler, twenty-six remained dry. In some cases pleurisy reveals itself by the discovery of high pressure on puncture without any fluid appearing, a condition regarded by Zink as due to pleural irritation without exudation (see Emerson's observations on p. 137). It is the writer's experience that a local pleurisy with thickening may occur if puncture is repeated too often at the same spot.

PLEURISY WITH EFFUSION

Pleural effusions in pneumothorax are conveniently divisible into three main classes :

CLASS I

Mild serous effusions which appear slowly, have little tendency to increase, and mostly disappear after a few

weeks or at most months. Among thirty-five exudates described by von Muralt fourteen, or 40%, belonged to this category.

CLASS II

Effusions which begin like Class I, but show a tendency to increase slowly with but few symptoms till a large quantity is present. Of von Muralt's 35 cases, 16, or 45.5%, belonged to this group. The fluid may last for years unabsorbed, and if it is removed it generally returns again. In a case of the writer's the pleural cavity had to be emptied four times before the formation of fluid began to abate. At the beginning the effusion is serous and lymphocytic, and tubercle bacilli are difficult to demonstrate. It may remain serous as in the case quoted above, or it may become purulent, either quickly within a few days, or slowly after many months. For its further course under these circumstances, the reader is referred to the heading *Purulent Effusions* on the following page, and especially to Jacot's Groups 1 and 2.

CLASS III

Pleurisies of acute course with high fever and general illness. Only 5, or 14.3%, of von Muralt's 35 cases were of this nature. Such pleurisies may follow infection elsewhere such as tonsillitis or "influenza," and the exudate tends to be richly cellular and may contain pyogenic organisms. Spengler observed five such cases in which diplococci were present in the exudate, but which, nevertheless, did just as well as cases of sterile effusion. Jacot's Group 3 of *Purulent Effusions* (p. 127) would also come under this heading, as would, indeed, direct infections of the pleural sac through perforation of the lung (p. 162), or by wounding of the lung with the pneumothorax needle, or from the outside during puncture, an accident which must be, in properly conducted operations, extremely rare.

Many writers, without remarking on their acute or

chronic nature, divide their effusions into serous and purulent. Thus Begtrup-Hansen remarks that of his 33 cases, 10 were purulent. Tecon and Sillig among 32 cases record 81·15% serous, 6·5% serous becoming purulent, and 12·4% purulent from the beginning. Since tuberculous effusions may be purulent or become so, this division gives no clear indication either of etiology or of the acute or chronic nature of the case; it only presents the matter from another point of view. Very useful and interesting, however, is Jacot's classification of purulent effusions which supplies an almost indispensable supplement to the preliminary division into three main groups.

Purulent Effusions.—Jacot was able to collect twenty-five cases for classification, and these he found to fall conveniently into four Groups:

(1) Pyopneumothorax of benign nature, always tuberculous in origin, beginning as a serous pleurisy (Class II, above) in a pneumothorax patient of good general condition. After the initial fever the temperature falls to normal and remains there, the patient keeps well, but the fluid gradually passes through a lymphocytic stage to turbidity, and finally after three or more months into frank pus. There is no tendency for this to absorb and generally it needs removal at intervals which fortunately become longer and longer. In a case of this nature under the writer's care, pus formation has only begun to slacken after over a year's treatment, during which time no less than $6\frac{3}{4}$ litres altogether, or nearly $1\frac{1}{2}$ gallons of pus, swarming with tubercle bacilli have been removed and replaced by gas. The patient meanwhile has felt well, led a fairly active life, and steadily gains in weight. Katz mentions two such cases clearing up after repeated tappings. Nine of Jacot's cases, or 36%, were of this benign character. Their treatment should be by fairly frequent tappings and replacement by air or gas at pressures sufficient to prevent re-expansion of the lung. It may happen occasionally that the fluid becomes thinner in its later stages, but commonly there is a

tendency to the formation of membranes and shreds of fibrin. If the lung is suitably collapsed these will lead to its permanent encapsulation; if the pneumothorax is only partial, or some expansion of the lung has been permitted, fibrinous bands may anchor the lung to the chest wall, split up the cavity into a number of isolated compartments, and finally lead to re-expansion of parts of the lung, causing, in some cases, an exacerbation of disease within it (see pp. 133 and 142).

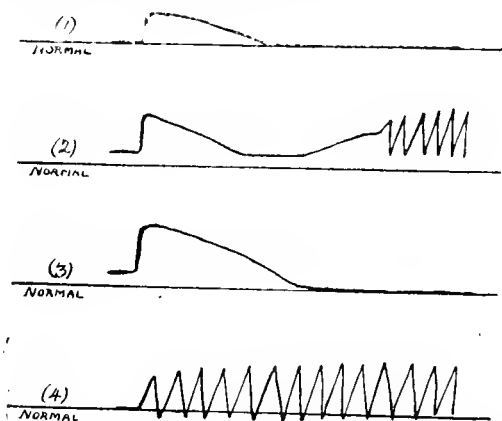


FIG. 10.—A DIAGRAM AFTER JACOT TO ILLUSTRATE THE TYPES OF FEVER ASSOCIATED WITH HIS FOUR GROUPS.

(2) Pyopneumothorax becoming grave secondarily. This occurs in pneumothorax cases which are febrile, and where the general condition is bad. The early stages are similar to those of Group 1, the fever of onset wanes and the fluid remains serous for two to seven months (Class II, p. 124). When, however, the purulent stage begins the temperature again rises and the case terminates in a hectic fever to which the patient finally succumbs after some months in spite of treatment. The picture appears to differ from that of perforation of the lung in the less sudden and violent onset of fever and the absence of pain in the side. Four, or 16%, of

Jacot's cases were of this nature. He has not found that a pleurotomy improves matters and only resorts to it where pus formation is very rapid, mainly to relieve pressure. He prefers, but without any expectation of recovery, lavage with lysol, Lugol's iodine solution, or collargol.

(3) Pyopneumothorax grave at first onset ; generally in cases of partial pneumothorax, still febrile and where the general condition of the patient is unsatisfactory. There is here no discoverable serous phase, but the onset with pain in the side and fever is precisely similar to that of a serous effusion save that the formation of fluid is more rapid. Treatment is by tapping and replacement with gas, and under these measures the fever drops gradually, often to a lower level than formerly, and the patient may do well in spite of the violence of the onset. Seven, or 28%, of Jacot's cases were of this nature, and of these five were cured within one and a half to ten months. In one case where he was tempted to perform a pleurotomy a fistula was still present one and a half years later. Jacot insists on the tuberculous nature of these acute cases which resemble the "septic" pleurisies in all particulars save in the absence of pyogenic microbes. He ascribes them to the breaking of a subpleural tuberculous focus into the pleural cavity.

(4) Jacot's fourth class is nothing less than pyopneumothorax due to perforation of the lung, a subject discussed in this volume under a separate heading (p. 162). Five, or 20%, of his cases came into this group, and all died of the empyema within two to ten months.

CHEMISTRY OF PNEUMOTHORAX EFFUSIONS

Work done on this line, mostly that of Mayer, shows that the effusions of pneumothorax come well within the designation of exudates. Thus the specific gravity falls above 1018, a figure commonly accepted as the lowest limit for inflammatory effusions, and the albumin

content is generally about 4% or over. Runeberg, as a result of his investigations of pleural fluids, found the following albumin content among different classes of effusion :

Inflammatory Exudates	.	.	4%—6%
Congestive Transudates	.	.	1%—3%
Hydræmia	.	.	below 1%

These figures must be taken to apply to freshly formed effusions, since transudates of long standing show a rise of albumin content when they result from chronic stasis or are under pressure. The same also applies to the exudates of pneumothorax cases. Mayer has shown, also, that there is more albumin in those going about or at work than in those confined to bed. Among his eighteen cases the albumin content was mostly near the minimal point for exudates, 4% or somewhat less ; it was found to increase with the gas pressure, to rise where there was no tendency to absorption, but to sink if absorption was imminent. It was highest in long-standing cases. All the cases gave Rivalta's reaction (precipitation with acetic acid), a test which hardly ever occurs in transudates but never fails in exudates. Two examples of clear effusions from among the writer's cases examined by Dr. Roodhouse Gloyne may here be quoted :

H.W..—Clear amber fluid with minute white deposit. Protein 3.5%. Cells chiefly endothelial, also a few lymphocytes and erythrocytes. Tubercle bacilli absent. Sterile on culture ; no antibodies. This was an effusion which rose 6 inches up the chest but was gradually absorbed by the end of six months.

Mrs. F..—Transparent amber fluid with small amount of flocculent white deposit. Reaction alkaline. Specific gravity 1024. Rivalta's test positive. Coagulable protein (dried and weighed) 5.375%. Tubercle bacilli absent. Sterile on culture. Antibodies present in small amount.

This effusion had to be tapped and replaced by gas on repeated occasions.

CYTOLOGY

Very few cellular elements are present save in cases of mixed infection where the albumin content is generally high and the cells polymorphonuclear. These cases apart, a lymphocytosis is characteristic and the longer the duration the more the cells. These "lymphocytes" are, according to Lippmann and Plesh, but offshoots of the serous endothelium and not of hæmatogenous derivation, and with this opinion Mayer agrees. In addition endothelial cells or plates are often found, evidences, according to Königer, of a traumatic irritation. In the fluid from Mrs. F. referred to in the preceding paragraph, the cell count showed polymorphonuclears 10·6%, small round cells 80·7%, and endothelials 8·7%. Eosinophiles are present in a large proportion of cases and are generally considered to indicate a favourable prognosis—10% or 12% was a not uncommon figure among Mayer's cases. It is in these cases especially that the presence of antibodies in the exudate is said to be demonstrable. Polymorphonuclear leucocytes may be present in large numbers in quite chronic tuberculous effusions, as in a case of the writer's where a sterile turbid fluid with a heavy deposit showed about 95% of these cells and numerous tubercle bacilli. In these cases, unlike acute infections, the pus cells tend to early degeneration and much débris is usually present. For figures of the proportions of purulent exudates likely to be met with the reader is referred back to p. 125.

BACTERIOLOGY

Apart from pyogenic infection—which may provide to view the pneumococcus, streptococcus, staphylococcus or other organisms—the question of bacteriology hangs round the presence or absence of the tubercle bacillus. As already remarked, Saugman found this organism in

all his larger exudates save only two. von Muralt's experience was somewhat similar with tubercle bacilli demonstrable, either microscopically or by animal experiment, in all examined, save in five acute cases. Begtrup-Hansen found tubercle bacilli in eighteen or nineteen cases examined. Mayer, on the other hand, only found them in about 45% of his cases—in ten animal experiment was negative, contrary to the results usually obtained for "idiopathic" pleurisy. Partly on the strength of this finding and also on account of other factors, Mayer has made a classification based on the character of the exudate, and this necessarily differs somewhat from the purely clinical classifications already put forward.

MAYER'S CLASSIFICATION

(1) **Exudates due to tuberculous pleuritis.**—Very highly albuminous with few morphological elements and often mixed at first but later becoming lymphocytic, especially if tubercle bacilli are sparsely present. No endothelial cells, eosinophiles few or absent. Resulting intra-pleural pressure not characteristic. Often exerts an unfavourable influence on the course of the case and associated with a spread of disease in the lung.

(2) **Exudates of acute infectious nature.**—After tonsillitis, bronchitis or other infections. Shows high albumin content, mixed cytology passing to leucocytic, or leucocytic from the beginning. Leucocytes much degenerated. Fleeting eosinophilia. Tubercle bacilli mostly absent. Intrathoracic pressure tends to rise, absorption is slow and case tends to do badly.

(3) **Exudates due to perforation of visceral pleura** (see p. 162).—The fluid is rich in leucocytes and these not degenerate but staining well. Endothelial plates, a few eosinophiles. Tubercle bacilli always present and often a mixed infection.

(4) **Exudates of a special type: the commonest form.**—Albumin abundant, 3% to 4%; Rivalta reaction

+ ; cytology spare and lymphocytic ; no endothelial cells. A high eosinophilia characteristic, up to 20%, and this accompanied by a blood eosinophilia. Specific complement-fixing antibodies, absent in the other types, are present in this, and exert a favourable influence. Tubercle bacilli are difficult to find, or absent. No increase of intrathoracic pressure. These exudates are of favourable outlook, especially where the eosinophilia is high or increases. They absorb quickly though they may reaccumulate without harmful results. Mayer ascribes them to the effect of nitrogen gas on a diseased and functionally disturbed pleura.

It will be seen that Class I of the clinical grouping is made up of cases of this type, Class II probably covers the remainder of Type 4 of Mayer's classification, and also includes Type 1. Class III consists of Types 2 and 3 described by Mayer. The classification of Mayer, though based on all too few cases, is suggestive and stimulating, and well worthy of further verification on a larger material.

EFFECTS OF PLEURISY IN PNEUMOTHORAX

On this point it is particularly difficult to generalise since a pleurisy, according to circumstances, may exert either a harmful or a beneficial influence, or perhaps none at all. Even in the individual case it may be difficult or impossible to prognose the effects of an effusion before these have already announced themselves. The points which work for good or evil, in these cases, may be separately considered.

Beneficial Effects.—The value of an effusion in the course of many cases of phthisis may be regarded as common knowledge, its mode of action being generally accepted as immobilisation of the lung. But it has been shown that these mechanical effects do not cover the whole ground. Königer's careful study of the question led him to remark that the beneficial effects might be present with minimal or fleeting effusions, and even

after an attack of dry pleurisy. On these grounds it has been held that, immobilisation apart, some serological influence must be exerted, and this view is supported by the improvement said to follow pleurisy in a proportion of cases of artificial pneumothorax where all mechanical effects are of necessity excluded. This view seems to fall into line with the discovery of specific antibodies in the fluid in many cases, as reported by Mayer and also by von Muralt for the effusions of pneumothorax. An immunising effect may proceed from these similar to that aimed at by Débove's "Auto-sero-therapy," but of slow and long-continuing effect. A general improvement accompanying or following the advent of an effusion in artificial pneumothorax belongs to certain cases only, and according to Mayer (see p. 131) is the special attribute of a specific type of case. The writer has not been convinced that any lasting influence, at any rate a favourable one, was exerted by pleural effusions among his cases. The only possible exception to this was a patient with a tuberculous empyema which persistently recurred after removal, the patient meanwhile putting on weight and appearing to improve in general health. A convenience, which may prove an advantage, following the occurrence of effusion in pneumothorax cases, is the marked reduction in the absorption of gas from the pleural surface. This allows of longer intervals between the refills, and removes the risk of lung expansion where the intervals, as often happens, tend to be longer than they really should be. It is quite probable, in the writer's opinion, that some of the improvement noted after an attack of pleurisy is due as much to this factor as to the serological influences already outlined.

In certain cases of partial pneumothorax, a mechanical advantage may accrue from the development of an effusion. In the first place adhesions may, it is said, be broken down by the pressure of the fluid and a more efficient cavity result. In the second place a stiffening of the mediastinum may allow of higher and more

effective pressures being employed than were permissible before the pleurisy occurred.

Unfavourable Effects.—The serious effects of a pyogenic infection of the pleura will not be considered here—they will be found under the heading of “Perforation of the Lung.” But, these apart, the pleural effusions occurring in the course of artificial pneumothorax are by no means always of beneficent influence, and the patient’s general condition may very seriously deteriorate. In effusions accompanied by fever and general symptoms, a temporary set-back is the rule, and this is often completely made up again, but in some this is not the case. This occurs particularly where, as not infrequently happens, a tuberculous pleurisy is accompanied by a spread of disease in one or other lung. Such a spread may show itself by a hæmoptysis, by an increase of sputum or accession of fever, or by the appearance of fresh physical signs.

Not the least serious of the effects of a pleurisy may be that on the final expansion of the collapsed lung. Thickening of the visceral pleura may prevent or greatly limit this expansion; if expansion is attained, on the other hand, the likelihood of adherent pleura is probably greatly increased. An occasional but most serious outcome of pleurisy is the gradual obliteration of the pneumothorax cavity under the irresistible pressure of contracting new fibrous tissue in the organised pleural layers. The effects of this pleural thickening, a true pleuritis deformans, do not come into play at once, but gradually a marked contraction of the side and drawing in of mediastinum and diaphragm come about as the fibrous tissue condenses. No gas pressure serves to delay the effect and a curious result commonly experienced is a drawing out and partial expansion of the collapsed lung towards the sinking chest wall. Such a lung expansion, unpreventable by the injection of gas, may lead to opening up of cavities and caseous areas, and a fresh lighting-up of disease. The risk of such serious after-effects of pleurisy is a strong argument

against the employment of pneumothorax therapy in cases still curable by ordinary sanatorium or other treatment, though it must be admitted that the occurrence of pleurisy is less common in cases of slight and early disease.

SYMPTOMS

Acute cases apart, pleurisy in pneumothorax may show but few symptoms to mark its onset. It may discover itself by the appearance of an unaccustomed intrapleural pressure, or the patient may announce the presence of a "splash," or this may be discovered by the physician.

W. N. announced a "splash" on moving on July 27th, and fluid was present to physical examination. This had been preceded by no symptoms, and no fever appeared till August 3rd. There was no rise of intrapleural pressure and all signs of fluid had disappeared a month later. This is a fairly average example of a pleurisy of Class I.

In some cases an unaccustomed intrapleural pressure from diminished gas absorption appears to be premonitory of the appearance of fluid, and this latter not discoverable till a later examination.

H. W. produced T.B. in his sputum for the first time on August 4th. A slight rise of temperature occurred between September 6th and 9th, and he lost some weight. On September 28th, his initial pressure $+2+11$ showed that but little gas had been absorbed during his usual interval, and only 50 c.c. of N were required in place of the usual 500 c.c. Nevertheless no signs of fluid were present, this being first discovered some weeks later.

Pain in the side and some amount of fever occur in the largest proportion of cases. The pain is generally in the side or back at about the level of the diaphragm, and is probably produced by a rubbing of the surfaces in the pleural reflection at this point. It may, on the other hand, be referred to the region of the shoulder (see p. 78). It may precede the appearance of fluid by many weeks.

Mrs. F. complained of pain in the left side on July 10th accompanied by slight morning fever, headache, and general aches over the body. The pain was accentuated by deep breathing and by movements of the arm, and there was tenderness on deep pressure. No increased intrapleural pressure or diminution of gas absorption occurred till August 7th, when a small amount of fluid was for the first time present.

Pain is not often severe and is generally relieved by aspirin and the application of warmth to the chest wall.

Fever also tends to precede the appearance of fluid, and unless it is associated with pain in the side, its significance may be at first doubtful. On the other hand, fever may not appear till after the fluid is present, and may be very slight and fleeting.

W. N., already quoted, showed a splash on July 27th, but no fever till August 3rd, when the temperature rose at night to 99 or 99.5 during ten days' duration, the fluid meanwhile increasing a little.

Fever may be considerable in some cases, as 101° or 102° or even 103°, and yet only an inch or two of exudate follow. The persistence of fever for many weeks does not afford any indication that the fluid is becoming, or is likely to become, purulent. In some cases the temperature may drop and remain normal, and the patient feel well and comfortable, and yet the effusion steadily increase from week to week.

Mr. C., after a violent onset with vomiting and high fever, produced only 1 inch of exudate which remained at this level during six weeks or more; the patient by then felt recovered from his attack, but the fluid without further fever or other symptoms steadily rose to 7½ inches in the ensuing six weeks.

There seems to be a marked tendency of the pleurisy, once an exudate is present, to light up again on but slight provocation. The writer has found a nasal or bronchial catarrh, or the shaking of a journey to be apparent causes of exacerbation in some of his cases. Exposure to cold is made much of by some authorities, notably Forlanini and von Muralt, and it seems possible

that the pleural surfaces may be more susceptible to such an influence where they are separated, as in pneumothorax, by a layer of gas. On this account it is wise to adopt particular precautions against chill in the case of pneumothorax patients living under sanatorium conditions.

Nausea and dyspeptic symptoms may usher in the onset of effusion particularly, according to some, when this occurs on the left side. A right-sided pneumothorax case of the writer's vomited at the onset of a pleurisy and continued doing so two or three times daily for the following three or four days.

The occurrence of pleurisy in pneumothorax is often suggested to the physician by the appearance of the patient. The face becomes pale and generally assumes that sallow yellow tint so highly characteristic of pleurisies of whatever nature. During the early stages, if fever and symptoms are present, the patient visibly loses weight and strength. When these have subsided the general health will again improve, though the fluid remains or even increases a bit. The writer has been impressed by the frequency with which pleurisy is associated with a pulmonary exacerbation. In such cases improvement will often quickly set in, but in bad cases it may mark the beginning of a serious or even fatal spread of disease. This was so in two of the writer's patients, both cases of advanced disease. In other cases such incidents as hæmoptysis, an increase of sputum, or the sudden appearance in it of tubercle bacilli, or the appearance of fresh signs, have preceded or accompanied the onset of pleurisy, and proved the more widespread nature of the attack. In many of these cases recovery does not appear to have been prejudiced by the temporary lung activity.

EFFECTS OF EFFUSION ON MANOMETER READINGS

These changes, due for the most part to a diminished absorption of gas, a lowering of elasticity, and stiffening

of the mediastinum, are best brought out by a few case references.

J. C.

Before onset of pleurisy	5 - 1	640 c.c.	+ 5 + 6
Pleurisy began	26.7.15		
6.8.15	. . - 0 + 4	100 c.c.	+ 4 + 7
18.9.15	. . + 1 + 4	35 c.c.	+ 4 + 8
15.12.15	. . - 4 + 1	30 c.c.	+ 5 + 9

These readings appear to show an increasing loss of elasticity so that enormous changes of pressure were produced by minimal amounts of gas. On these occasions but one or two inches of fluid, tuberculous pus, was demonstrable, but that part of the diminished elasticity was dependent on the actual fluid layer appears to be shown by a gain immediately after the fluid had been replaced by gas thus :

2.6.16	. . - 4 + 2	150 c.c.	+ 5 + 8
--------	-------------	----------	---------

rs. F.

Before onset of pleurisy			
23.7.15	. + 1 + 6	400 c.c.	+ 7 + 10
With 1 inch of fluid			
6.9.15	. + 1 + 6	140 c.c.	+ 7 + 11
25.9.15	. + 11 + 15	gas removed	+ 6 + 10

Here again the presence of fluid reduced the elasticity, and in addition a decided rise of pressure occurred without any increase of fluid, suggesting an actual contraction of the space brought about by irritation of the pleural surface.

In this connection reference must be made to the interesting experiments of C. P. Emerson on the accommodative powers of the pleural cavity. He found that the chest, by elevation of the ribs and descent of the diaphragm, can accommodate very various quantities of fluid without any change of pressure. If fluid is continuously injected into the pleural space, the pressure, of course, must rise, but it tends to do so in stages or jerks, owing to attempts on the part of the chest to accommodate itself to the increase and so keep down the pressure. This he ascribes to a reflex mechanism since

his experiments were performed on dogs anæsthetised with ether, and some disturbance of this mechanism may well afford explanation of certain of the curious pressure anomalies of hydro-pneumothorax, such as that just referred to, and even of the changes ascribed above to loss of elasticity.

H. W.

Before onset of pleurisy

31.8.15 . - 7 - 4 740 c.c. + 3 + 11

14.9.15 . - 5 + 5 520 c.c. . + 7 + 14

After onset of pleurisy

28.9.15 . + 2 + 11 50 c.c. + 2 + 13

This demonstrates diminished absorption of inflamed pleural surfaces. No fluid was demonstrable till some weeks later.

Weiss has remarked that the respiratory fluctuations are diminished in the presence of pleural effusion, but this has not been the writer's experience. Thus for example :

J. C.

		Respiratory fluctuations.	
		Before filling.	After filling.
Before pleurisy	.	4 cm.	2 cm.
	.	4 cm.	1 cm.
	.	4 cm.	2 cm.
	.	4 cm.	1 cm.
With fluid.	2 inches up .	4 cm.	2 cm.
" "	7½ " " .	4 cm.	3 cm.
" "	4 " " .	5 cm.	5 cm.
" "	6 " " .	6 cm.	4 cm.

PHYSICAL SIGNS

Dry pleurisy may give but little evidence of its presence, since no rub can appear unless at the diaphragmatic reflection. In one of the writer's cases fever and chest pain were followed by the appearance of an area of light impairment over part of the chest wall, suggestive of the deposition of pleural lymph though no fluid appeared. The writer has noticed areas of similar slight impairment round the spot where repeated punctures have been made, in cases where the increasing depth of puncture needed suggested the development

of pleural lymph. Such pleural membranes may be pushed before the needle with the creation of a false pocket—a painful and somewhat puzzling mischance. The X-rays will afford some help in the detection of pleurisy without effusion.

Effusions.—The presence of a small quantity of fluid is easier to diagnose with pneumothorax than where this is absent. The “succussion splash” will probably detect even a few drachms of fluid if the stethoscope is applied close to the base and the proper impetus given. For this purpose the chest should be held between the two flattened hands, the stethoscope end being grasped between the thumb and index finger of one of them, and thus applied to the chest wall. Anything more than a trace of fluid will give an area of movable dulness at the base, and in gauging its amount it must be noted that the pleural cavity at the back of the chest (and the resonance in pneumothorax) extends fully 1 inch lower than the lower margin of the sound lung. Thus with an inch of fluid, resonance extends to the same level on the two sides behind.

With effusions larger than a trace, certain points determined by the presence of gas are noteworthy. The movable nature of the fluid has been already referred to. In addition the parabolic curve so characteristic of the upper level in ordinary pleurisies is lost, at any rate in the larger effusions. In small effusions the writer has noted a modified parabolic curve, but in large effusions the upper limit is horizontal. Corresponding to this is the loss, to radiological examination, of Garland’s triangle produced by lung retraction allowing the fluid to creep up to the highest point in the axillary region—the upper line is horizontal to X-rays as to physical examination. Dulness to percussion is similar to that of effusions generally, though the writer thinks it is less resistant and certainly the upper limit is harder to fix with accuracy. In place of Grocco’s triangle at the opposite base the writer has generally found an area of quadrilateral outline; this may also be observed in

pneumothorax apart from the presence of effusion (see fig. 5, p. 85). In some cases, it is said, a mediastinal hernia may convey the impression of a double-sided effusion—a warning, indeed, against harmful intrapleural pressures.

To auscultation the feeble breath sounds of pneumothorax may alone be noted. In many cases, however, the fluid appears to conduct sound better than the gas above it, and loudish bronchial breathing and friction may be audible over the fluid, the friction, presumably, being produced at the extreme limit of the pleural reflection. In some cases signs of this nature may point to re-expanding lung. Where the lung cupola is adherent, bronchial breathing and crepitations may appear at the apex above the effusion, and point to lung congestion or consolidation, a not uncommon accompaniment of the larger effusions. These signs are readily conducted to the opposite side, especially in the interscapular region, and may lead to a very confusing clinical picture. Vocal resonance may be nasal and somewhat diminished with these pleurisies while vocal vibration, often quite good over the pneumothorax above, is lost over the fluid. Mayer has described tympanitic areas over the fluid caused by the presence of tongues of adherent lung; these, if found, must be carefully avoided on puncture.

To X-Rays the fluid takes a horizontal level as already mentioned; the displacement of organs may be more or less according to the intrapleural pressure. In some cases fluid may be held up by basal adhesions or may be at first interlobar, conditions for whose discovery the X-rays are better adapted than are physical signs. Saugman and Hansen regard the appearance of paradoxical diaphragmatic movements, Kienbock's phenomenon, as an early sign of effusion.

TREATMENT

The treatment of effusions in pneumothorax cases should be made as conservative as circumstances will

allow—it must necessarily differ according to the nature of the individual case. At the first appearance of symptoms suggestive of pleurisy the patient should be sent to bed, warmth applied to the side, and salicylates given. According to Saugman these latter exert a specific effect.

Chronic Effusions.—Small serous effusions should be left alone, the pressure being taken at intervals and kept at about the accustomed figure by the occasional addition or removal of gas. The same measures apply to larger chronic effusions, but in these particular care must be taken lest the lung expand under the fluid and become adherent to the chest wall. Patients are often quite comfortable with a considerable bulk of fluid in the pleural cavity and some gas must always be kept above the fluid so that the lung remains under an effective positive pressure. This pressure may have to be raised if the lung gives evidence of re-expansion. If the fluid causes respiratory distress, particularly on the right side by hampering the thin-walled right heart, or if it threatens to fill the whole pleural cavity, it must be withdrawn and replaced by gas (see Gas Replacement, p. 145). This operation may have to be repeated several times, as in two of the writer's cases in which the pleural cavity has had to be emptied on four and five occasions respectively up to date. When the fluid has ceased to increase attention must be directed, especially by radiological examination, to keeping the lung collapsed by the needful refills at the same or an increased pressure, the patient meanwhile in many cases being able to go about as usual. Mayer recommends the trial of Débove's "auto-serotherapy," the subcutaneous injection of 1 c.c. of the exudate immediately on its removal, in the treatment of serous effusions. In cases where specific antibodies are present a reaction is said to occur in six to eight hours after the injection with fever and other symptoms similar to those experienced in a tuberculin reaction. Diuresis follows, and at the end of two or three days the pleural fluid has gone.

von Muralt hesitates to approve of this treatment owing to the fact that virulent tubercle bacilli may be present in the pleural fluid. The larger effusions may remain unabsorbed for months or even years, and in many cases appear to do no harm even though the fluid is turbid or quite thick and contains numerous tubercle bacilli. If pleurisy occurs in a small partial pneumothorax, there is danger lest the cavity be obliterated and the lung drawn out by the action of organising lymph and spreading adhesions. For the avoidance of this the pressure must be still further raised (it has generally been high in these cases), and in some the fluid removed if not too thick to pass through a needle. But organisation of lymph and fibroid contraction of the pleural space is a danger which may also threaten a complete pneumothorax under the influence of a chronic pleuritis (see p. 133). The fluid becomes thicker, the lymph masses organise and the collapsed lung becomes embedded in a mass of fibrous tissue. This has generally occurred, in von Muralt's experience, in cases of advanced lung disease and provides in such cases a not altogether unsatisfactory termination. In cases, however, where the other lung might need collapse-treatment at a later stage, such an occurrence must be warded off by the replacement of fluid by gas at an early stage, and Spengler and Sauerbruch recommend this measure for all effusions which show no tendency to absorption. Thereby, also, the risk of the lung expanding under the fluid and becoming adherent to the chest wall is avoided. Examples of forcible re-expansion of an unhealed collapsed lung through fibroid contraction of a pneumothorax cavity, with resulting exacerbation of pulmonary disease, are fortunately rare; should this occur some form of plastic operation offers the sole remaining chance of obtaining further collapse of the lung.

Acute Serous Pleurisy.—In effusions accompanied by fever, intolerance of the accustomed intrathoracic pressure may be evident, and the fever tend to drop only when this is reduced to atmospheric level. The

lowering of a high pressure may be accomplished by merely releasing gas through the tap of the pneumothorax needle, but if the pressure is to be brought as low as the atmospheric this should be done by lowering of the pressure bottle so that gas is sucked back into the nitrogen chamber. It is important to remember that the tubing and filters must be re-sterilised if this manœuvre has to be carried out with the ordinary pneumothorax apparatus. If, in acute and febrile cases, thus lowering the pressure does not remove the symptoms, the whole effusion should be drawn off and replaced with gas (see Gas Replacement, p. 145) without waiting for a reduction of activity. This commonly leads to a drop of fever and general improvement. Forlanini replaces with air instead of nitrogen in these cases, the choice of gas depending on its desired rate of absorption. The fluid may return once after removal or even twice, but very rarely, it is found, for a third time in these cases. Where effusion is accompanied by fever and other symptoms, it must be ascertained that these are not due to an underlying lung exacerbation before treatment directed to the pleural condition is undertaken.

Acute Purulent Effusions.—These, whether tuberculous or the result of a mixed infection, must be treated conservatively no less than serous pleurisies, since if the pleural cavity is freely opened to the air the patient will be left with a suppurating cavity impossible to fill and close (see Perforation of Lung). At the outset the fluid should be removed and replaced by air or N. This, repeated if necessary, should suffice in the case of a tuberculous effusion (see Jacot's Group (3), p. 127). If no improvement follows this measure in the presence of a mixed infection, the fluid should be again drawn off and the cavity washed out through the pneumothorax needle with some litres of $\frac{1}{2}\%$ to 1% lysoform solution, continued until the fluid returns clear through the aspirating needle. This manœuvre, first recommended by Forlanini, is highly commended by Spengler and Sauerbruch, who have found that most cases proceed to healing under

this treatment. Dumarest uses warm lysol solution up to 5% passed through in small quantities. The injection of such antiseptics as quinine, collargol, and iodoform preparations appears to achieve but little good in these cases, though von Muralt has seen improvement in small effusions so treated with electrargol. If a thoracotomy becomes eventually necessary this should be preceded, according to L. Spengler, by constant draining so as to reduce the cavity by lymph formation to the smallest possible dimensions before opening. In cases resulting from lung perforation the chances of saving the patient's life are sadly remote since the perforation in the collapsed lung can seldom be made to heal. Spengler has, however, cured some cases by repeated plastic operations to reduce the size of the thorax until the wound was eventually closed (see *Perforation of Lung*).

CHAPTER XII

GAS REPLACEMENT

THIS operation was first performed by R. W. Parker, in this country, in cases of empyema, and described in a paper read before the Medical and Chirurgical Society as early as April 1882. It was utilised, independently, by Potain two years later in the treatment of effusions complicating spontaneous pneumothorax. At a still later date Vaquez, Achard and others applied it to serous effusions, and in recent years it has again been recommended by Küss and Wenckebach in the treatment of certain cases of empyema. Since gas replacement is occasionally needed in pneumothorax cases and has been referred to in foregoing paragraphs, some description of the procedure seems called for here.

Instruments needed.—(1) Those already noted as required for a gas filling (p. 43).

(2) Potain's aspirator and trocars. The apparatus should be fixed up before the operation and water aspirated through it so as to establish its efficiency. This is especially important because the pump usually supplied with this instrument is fitted to act, through different openings, either as exhaust pump or force pump. The use of the force pump by accident is likely to be followed by disastrous consequences.

(3) Collodion dressings and adhesive plaster.

The Operation.—The patient should lie flat while certain landmarks are pencilled, notably the border of the displaced heart and the usual lower level of the lung, or the lowest limit of gas resonance, *i.e.* the limit of the

pleural reflection; in all chest operations it is well to establish this graphic warning of the presence of surrounding organs. The patient then turns towards the sound side with the arm raised and the aspirating trocar is entered about the posterior axillary line or somewhat behind this at a level about that of the lower lung border or somewhat above this. In cases of effusion the diaphragm, owing to the weight of fluid and the gas pressure above it, is displaced considerably downwards so that the trocar might be entered much lower and still strike the pleural cavity. There is, however, no advantage in this, and also a possible disadvantage, since, if the pressure is unduly lowered, the diaphragm may rise and obstruct the trocar, an accident whose explanation might not be at once understood. When the trocar is in place, the patient is turned back so that the point of aspiration occupies a dependent position. The trocar is now withdrawn and a little fluid sucked into the aspirating bottle to determine that the cannula is in place.

The pneumothorax needle is now entered in front at a point where good resonance guarantees the presence of gas. This is best accomplished at the base of the chest, the patient's head being lowered so that the gas accumulates in this region—the chest wall is thicker to perforate near the apex and adhesions more likely to be entered. As soon as the position of the needle in the pleural cavity is established by the usual manometer fluctuation, the pressure is recorded and the removal of fluid begins. The aim should be to maintain the patient's usual positive pressure as near as may be throughout this operation. If the initial pressure is high, some fluid may be released before the gas bottle is opened, but afterwards it is, in the writer's experience, best to keep the gas flowing in at an even pressure all the while the fluid is running out. This is accomplished by gradually raising the pressure bottle as the gas flows, so as to maintain, as recorded by the manometer, the same positive pressure, perhaps some 5 or 6 cm. H_2O mean

pressure. This, the ideal method, can only be carried out where the movable bottle type of pneumothorax apparatus is used. Those who work with fixed bottles recommend, as does Forlanini, the alternate flow of fluid and gas, 100 c.c. to 200 or 300 c.c. at a time, a somewhat tedious procedure where 2,000 or more c.c. have to be displaced. A case of the writer's which he displaced partly by this alternate method showed the following readings :

Gas put in.	Fluid Removed.	Manometer reading.
		+ 2 + 6
	200 c.c.	- 4 to 0
150 c.c.	.	+ 4 + 8
	200 c.c.	- 4 to 0
200 c.c.	.	+ 4 + 8
	200 c.c.	- 4 to 0
200 c.c.	.	+ 6 + 9
	300 c.c.	- 3 to 0
200 c.c.	.	+ 2 + 6
	230 c.c.	- 8 - 2
470 c.c.	.	+ 4 + 8

Here it will be seen that considerable alterations of pressure were produced, and though these might have been reduced by the replacement of only 100 c.c. each time, yet the continuous displacement is admittedly more perfect and seems to the writer a further argument in favour of a movable-bottle pneumothorax apparatus. The removal of fluid in any quantity without its replacement by gas is, of course, not to be considered in any pneumothorax case. There is no longer a displaced lung to take its place, as in an ordinary pleural effusion, the fully collapsed lung (perhaps fortunately) will not readily expand, and aspiration of fluid soon develops a high negative pressure for which a harmful displacement of the mediastinum and rise of the diaphragm will attempt to compensate. This is, of course, a very undesirable result likely to lead to pain and distress, and if the lung expands, possibly to more serious symptoms. Thus Weiss, who aspirates to a negative pressure causing cough, dyspnœa, chest pain, and inspiratory retraction in

the supraclavicular region, before he replaces with gas, finds that the operation is nearly always followed by a rigor and fever above 39° C. (102.2° F.) of several days' duration, a result which can hardly be considered surprising.

When the fluid has all been removed gas and froth begin to bubble out through the trocar, and this should now be removed. In doing this the puncture must be carefully sealed with gauze and collodion, and over this a pad may be strapped. Leakage into this needle track is very apt to cause an inflammatory swelling or even an abscess and sinus.

The gas pressure is now adjusted to a suitable figure, and this is usually positive but somewhat below the usual since it is more likely to be augmented by the secretion of further fluid than lowered by absorption of gas. Forlanini meets this difficulty by replacing with air (see remarks on p. 33). If the operation has been skilfully performed the pneumothorax side should be resonant even down to the limits of the pleural reflection 1 inch below the lower margin of the opposite lung in the dorsal region. It is interesting that the amount of gas required seems always somewhat less than that of the fluid displaced, even allowing for differences of initial and final pressure. This seems to the writer due to the greater displacement of diaphragm and lower levels of the mediastinum by the fluid on account of its great weight, these structures recoiling somewhat under the lower pressure of the elastic gas. In a case of the writer's 1,630 c.c. of fluid was replaced by 1,420 c.c. of gas, and in another, on three occasions, 2,500 c.c. of fluid by 1,830 c.c. of gas, 1,400 c.c. of fluid by 1,350 c.c., and 1,450 c.c. by 1,250 c.c., to bring the intrapleural pressure up to the same figure.

The effect on the patient of a gas replacement properly performed is quite insignificant, being little more than that caused by an ordinary nitrogen refill; the operation is very much less serious than an aspiration alone on account of the avoidance of changes of intrathoracic

pressure and displacement of organs. Indeed, in an ambulant pneumothorax case the operation can be quite safely carried out in the consulting room, and the writer has done this on many occasions, the patient afterwards returning home in a taxicab under the care of his medical attendant. This could, of course, never be done with safety in the case of an ordinary pleural effusion.

It has happened in some cases that a fistula develops in connection with the puncture wound. Weiss and Brauer have each had a case of this, and the latter remarks that, if secondary infection is avoided, the fistula closes again after some months. The writer has experienced subcutaneous inflammatory swellings the size of a tangerine orange at the puncture sites in a case with chronic purulent effusion containing abundant tubercle bacilli. These were reminiscent of excessive local reactions to tuberculin and remained over long periods, and even proceeded to definite fluctuation. One of these cold abscesses after a period of three months suddenly acquired a pneumococcus infection and became acute, but fortunately no further trouble occurred after this was opened, and no sinus formed.

In cases of effusion in pneumothorax it may happen, as on two occasions to the writer, that the whole pleural cavity has become full of fluid before the case presents itself for aspiration. This must be avoided when possible, since the operation is more troublesome where no adequate gas bubble can be found for the pneumothorax needle to enter. The conditions are then similar to those of pleural effusion apart from pneumothorax where a replacement with gas has been decided upon. In such cases a needle of wide bore is indicated, such as the writer's trocar and cannula depicted on p. 34, since otherwise pressure fluctuations may fail to be communicated to the manometer through the fluid layers. Where this is the case anxiety will be felt lest the needle has entered an adhesion, an accident particularly warned against by Mayer in these cases. In the absence of a manometer fluctuation evidence that the end of the needle

is in the pleural cavity may usually be obtained by the appearance of fluid in the manometer tubing; a small length of glass tubing near the needle should form part of the usual apparatus, so that this may be visible. Failing its appearance a syringe, after the pattern of the "safety" syringe of Forlanini, may be applied to the end of the needle, and fluid aspirated into it. The position of the patient may be moved to facilitate this test if it does not come off at the first trial. As soon as fluid appears the intrapleural location of the needle end is established and gas may be entered while fluid is withdrawn just as already described. The gas tubing should be kept raised so that fluid cannot run far up it and the continuous passage of gas will usually suffice to dry the needle, so that proper manometer readings are available before the end of the operation.

CHAPTER XIII

THE ACCIDENTS OF PNEUMOTHORAX TREATMENT

PLEURAL SHOCK

PLEURAL shock presents a series of symptoms which are generally indistinguishable from those caused by gas embolism (see p. 156). On this account difference of opinion as to the cause of symptoms arising in individual cases is bound to occur, and cases of embolism have probably been from time to time described under the heading of pleural reflex or pleural shock. But in spite of this there seems no doubt that pleural shock is a reality, and the fact that it tends to repeat itself time and again in the same patient suffices to exclude embolism in a proportion of cases.

Symptoms.—The symptoms have appeared as a rule with great suddenness at the moment when the needle is entering or leaving the pleural space. More rarely they come on after an interval of fifteen to twenty minutes, or even after some hours.

In a typical case the patient develops a sudden pallor, loses consciousness and soon becomes cyanosed. The pulse and respiration become irregular, the pupil large, and clonic and tonic spasms of the limbs ensue, or these occur only on the side of the pleural reflex or in isolated muscles. Some patients cry out at the onset. Forlanini divides the symptoms into three groups:

Psychic Symptoms.—Giddiness and loss of consciousness.

Motor Symptoms.—Tonic and occasionally clonic spasms, flaccid paralysis with contractions, hemiplegia or monoplegia. Never involuntary micturition, seldom vomiting.

Circulatory and Respiratory Symptoms.—Rapid, weak, irregular pulse, pallor of skin, cyanotic patches over face, neck and thorax.

It is impossible to say amid this medley of symptoms what, if any, have been drawn from cases which were in reality embolic. De Cereville thinks that a flaccid paralysis points to embolism and not to pleural reflex, of which, also, a much more acute disturbance is characteristic. His experience of shock symptoms, the result of irrigation of the pleural cavity, include the appearance of unequal pupils, convulsions, coma, and sudden death. Occasionally he has seen vaso-motor disturbances—general skin flushing, retinal hæmorrhage. Mere contact of a probe with sensitive granulations sufficed to bring about symptoms in some cases. Symptoms of shock last a few minutes up to half an hour or even for hours (Forlanini), and no memory of the attack remains after recovery of consciousness.

Conditions of its Appearance.—It seems a peculiarity of pleural shock that it is apt to reappear at subsequent fillings, and to get worse and even become fatal if treatment is persisted in (Forlanini). Forlanini remarks that if it occurs twice it is best to give up the treatment. In 134 cases with over 10,000 fillings he met it in 12 cases, in 5 of whom it was repeated and treatment abandoned. Other authorities have had much less experience of shock, and the main description of this condition comes from the writings of Forlanini.

Saugman sounds the warning that we must only speak of shock when embolism can be excluded, and Brauer is even more emphatic on this point. Saugman during seven and a half years of pneumothorax treatment had 22 accidents altogether, of which 6 were mere faints. From December 1906 to October 1910, when his first fatal accident occurred, he treated 98 patients

with 2,200 punctures and experienced 14 accidents. In those days he was careless of puncturing the lung. From October 1910 to September 1912, when his second fatal accident occurred, he treated 63 fresh patients with 1,200 punctures and only 2 accidents. During this period he exercised great care to avoid wounding the lung. Since September 1912 he had treated (up to date of communication in 1914) a further 49 fresh cases with 2,000 punctures and no accidents. It is difficult to say how many, if any, of these accidents could be ascribed to pleural reflex. He thinks shock greater where blunt instruments are used—he is also careful to avoid more than two punctures at a first operation. If shock occurs he desists from further operation unless an open incision after Brauer's method. Sachs records 26 cases of pleural shock, none of them fatal, among 1,058 pneumothorax cases from the American literature, a percentage incidence of 2·5. Lillingston saw slight shock on only one occasion among 32 patients. Brauns experienced pleural reflex with fainting twice in one nervous patient. The writer with many hundreds of fillings up to date has had no experience of shock, though he uses a blunt instrument at all first operations. De Carle Woodecock and J. A. M. Clark have noted that shock symptoms are much more commonly seen in cases of early disease where the pleura is healthy, than in cases of more advanced tuberculosis—a strong argument, in the writer's view, against the treatment of early phthisis by artificial pneumothorax (see p. 19).

It is reasonable to expect, if pleural shock is a real entity, that many cases must have been recorded during various surgical manipulations of the pleura in the past. This is the fact, and Zesas has been able to collect from the literature records of 54 cases where serious pleural shock occurred. Twenty-one of these were of fatal issue, but 33 recovered; all were in cases of empyema. Thirty-two occurred during irrigation of, and injections into, the pleural cavity; 14 during empyema operations; and 8 during explorations. In 45 the clonic and tonic

convulsions came on at once, but in 8 after some minutes or even hours. In 3 patients they occurred on several occasions. In some mere touching of the pleural surface with a probe or drainage tube sufficed to elicit symptoms. In these the evidence for pleural shock seems overwhelming. In some others, where the manœuvre which led to symptoms had been previously practised, often many times, without result, the explanation seems more doubtful. In such, a difference of temperature of the lotion or injection-fluid might afford an explanation, and this was recorded by Paget and by Cayley in a couple of cases. But the possibility of embolism, whether of blood clot or gas, cannot in these cases be excluded. Indeed its occurrence seems probable in that paresis or paralysis of one or more limbs, or complete hemiplegia with disturbance of speech, is described as the after-effect in a proportion of cases.

In animals it has been shown that mere mechanical irritation of the pleura will not cause shock, but it is readily produced by the introduction of chemical irritants such as tincture of iodine, phenol, acetic acid, and alcohol. Cordier showed by experiment that shock occurred mainly through the vagus nerve—but he also found that ligature of the carotids prevented its occurrence, and that when the ligature was cut and the cerebral circulation re-established after two or three minutes, convulsions and death ensued. Cordier discovered, in addition, that shock could be avoided with certainty if an anæsthetic was given at the time of the operation, or morphine beforehand.

Prophylaxis.—The treatment of shock is especially prophylactic and its most important point is based on the discovery of Cordier referred to above. In all cases an injection of morphine or omnopon (p. 42) should be given before the initial operation, and in some nervous and apprehensive patients it may be repeated before the first one or two refills. In addition the careful use of a local anæsthetic, of which novocaine appears to be the safest (pp. 43 and 44), giving time for it to act before each

forward movement of the needle, should reduce the risk of pleural shock almost to nil. Indeed in recent years, since precautions of this sort have become universal, one hears but little or nothing about pleural reflex. Other precautions consist in the avoidance of the menstrual and premenstrual period in women, and confining the first operation to two punctures according to the advice of Saugman. There is no doubt that the patient's nervous stability is visibly lessened by the strain of two unsuccessful punctures at a first operation. The writer has been accustomed to give a small dose of alcohol or sal volatile at the first operation in some nervous patients.

Saugman also advises that an assistant keeps a finger on the pulse during the initial operation, and that this be suspended if any irregularity or marked diminution of volume occurs.

Treatment.—If in spite of these precautions “collapse” symptoms should appear, their treatment must be prompt indeed if success is to be achieved; the necessary drugs and apparatus must be to hand in case of such symptoms arising. Since embolism cannot be excluded, a line of treatment appropriate, so far as possible, to either or both conditions must be adopted. The needle should be immediately withdrawn, the patient's head dropped as low as possible, and ether or ether and camphor injected. Saugman recommends faradisation of the heart region and of the phrenic nerve, rhythmic traction of the tongue, and finally the intravenous injection of digalen. Forlanini gives, in addition to restoratives, whiffs of ether or chloroform and performs artificial respiration. This last measure should, however, be confined to cases, such as those of repeated attacks, where gas embolism can be excluded with some certainty.

GAS EMBOLISM

Cases of gas embolism have been, as already suggested, often attributed in the past to pleural shock. As a matter of fact it has never been possible to disentangle the symptomatology of these two conditions, and there remain but few points upon which a differential diagnosis may be based during life. The exclusion of conditions which might have led to embolism is notoriously difficult, since the gas may come from the manometer tubing or, according to Brauer and others, from the alveolar air, when the needle has entered the lung.

Two points stand out above the mass of general symptoms as capable of giving the clue in an individual case—on the one hand the reappearance of symptoms at more than one filling is strong evidence of their reflex nature and points to pleural shock; on the other the discovery of gas bubbles in the retinal vessels, as established by Brauer and his co-workers Stargardt and Wever, is definite proof of gas embolism. Unfortunately these diagnostic points are not always available. Even in fatal cases with full postmortem examination the evidence of gas embolism may be doubtful or incomplete. Gas is normally present in the veins of the pia mater, but, this apart, the discovery of gas in the blood stream at any point may be held sufficient, in the opinion of Brauer, to establish a diagnosis of gas embolism.

Mechanism of Gas Embolism.—It seems fairly certain that the introduction of gas into the greater circulation, as by puncture of a vein in the chest wall, in the quantity and with the rapidity probable at a pneumothorax operation, is hardly likely to give rise to gas embolism. Nevertheless this accident happened to a case under the care of Zink. Such gas is carried through the right side of the heart into the pulmonary circulation and its passage to the brain is blocked. It is, then, the lesser or pulmonary circulation with which we must concern ourselves, since gas from a

pulmonary vein enters the left heart and may proceed straight to the cerebral circulation. In the absence of adhesions the lesser circulation is sharply cut off, and it is only by entering the lung that a pneumothorax needle can reach it. On the other hand, when pleural adhesions are present a wide communication between the two circulations may exist through the formation of new blood vessels, such vessels often becoming almost angiomaticous in character. But even when this condition exists it appears that the blood usually flows towards the greater circulation, and substances injected into the pulmonary veins appear in the intercostal and diaphragmatic vessels. To cause cerebral embolism it seems as if it must be necessary to wound the lung deeply, and this probably accounts in part for the rarity of this accident in recent years when the lung's integrity has been more scrupulously respected than in the past.

Intravenous Pressure.—The entry of gas into a wounded vein depends on the negative intravenous pressure, a force which is maintained both by the suction power of the thorax and, to a less extent, by that of the heart. The negative pressure in the intrathoracic veins lies but little above the general intrathoracic pressure and shows the same fluctuations during inspiration and expiration. Thus Piper in experiments on cats and dogs with a cannula passed down the jugular vein into the thorax found such pressures as -12 cm. H_2O during expiration, -14.8 cm. during ordinary inspiration, and -19 cm. on deep inspiration. These showed a mean difference, when cardiac fluctuations of pressure were taken into account, of about 5 cm. H_2O above the general intrathoracic negative pressure taken at the same operation, both during inspiration and expiration. But the minimum difference amounted to little over 3 cm. H_2O , and if these figures are at all applicable to man it is obvious that a very powerful suction power exists in the pulmonary veins, particularly during inspiration and the heart's diastole. Such a suction power might amount in man perhaps to -7 or 8 cm. H_2O

or even more, and in such an accident as blocking of a large bronchus in a case with adherent pleura, a very much higher negative pressure might be developed. The accidental entry of gas is perhaps most likely to occur where a vein is surrounded by disease which stiffens its walls and hinders its collapse.

The Source of Gas Embolism.—The lung being wounded there are only two sources, in a properly conducted operation, from which gas can enter a pulmonary vein—one is the alveolar air, and the other is the tubing leading to the manometer and gas apparatus.

Alveolar Air.—As to the first, its possibility is vouched for by such authorities as Brauer and Spengler, and has the support of Saugman. These authorities point out that the alveolar air is at a slight positive pressure, while the venous pressure is negative, especially during inspiration. Saugman has experienced even mediastinal emphysema from mere wounding of the lung, though here there could be no negative pressure to assist. Brauer had himself a case of fatal embolism during the use of a Paquelin's cautery whereby a vein only the size of an ordinary hat-pin was wounded. In this case gas bubbles were observed in the retinal vessels. Brauer remarks that this accident is especially liable to happen where the vein is stiffened by surrounding infiltration; he also insists that but a small volume of air may be required, and inspiratory suction, apart from the cardiac suction, may suffice to bring it about. On this account Saugman insists that the patient be warned against deep breaths and coughing during the early stage of the operation. Begtrup-Hansen has recorded a case with adherent pleura where the lung was wounded and symptoms began two or three minutes after withdrawal of the needle, coughing having led to fatal embolism. In a case of Mayer's a deep inspiration displaced the needle into an adherent tongue of lung tissue and a fatal embolism followed.

From the Manometer Tubing.—Gas from the manometer tubing seems a possible source of embolism,

particularly if such tubing and the manometer are of needlessly wide bore. Not only must the amount of gas displaced from one arm of the manometer by a rise of say 5 or 6 cm. be considered, but also the expansion of the whole volume of gas in the tubing and apparatus as a result of the negative pressure. This might supply a dangerous volume of gas in apparatus of wide bore. On this account and also for other reasons (see pp. 28 and 29) the writer advises strongly that apparatus of narrow bore, .3 or .4 cm. diameter, be used, but it is questionable whether even this affords complete protection against gas embolism if the lung is pierced or torn. Saugman, to avoid this danger, uses at the initial operation a water manometer close to the needle and of such bore that a fluctuation of -14 cm. allows the entry of only 1 c.c. of air or gas. This measure was devised and recommended by Würtzen.

Volume and Nature of Gas.—In considering the danger of gas embolism it is obvious that the volume and the nature of the gas are of importance. Forlanini, in operating on dogs, found that 6 to 8 c.c. of N could be put into the left ventricle, or 2 to 3 c.c. into the carotid, without symptoms of embolism resulting—more than this gave serious symptoms. Saugman considers that 20 to 30 c.c. are needed to produce fatal embolism in humans, but Brauer records a death after 15 c.c. of gas, though the diagnosis of embolism was unverified by post-mortem examination. Brauer holds the results to depend more on the part of the brain implicated than on the quantity of gas introduced. Many operators use O_2 at the initial operation on the recommendation of Deneke with the belief that it is quickly absorbed in the circulation and hence less likely to lead to embolism than is N or air. Brauer, however, points out that though O_2 is quickly absorbed in venous blood, and hence in the greater circulation, this does not apply to the pulmonary veins, the real source of danger, since in these the blood is arterial. Experiments carried out by him, with the assistance of Wever and Stargardt, on

rabbits, dogs, and apes, go to show that the kind of gas matters little, and that embolism occurs alike with O_2 , with air, or with N.

Symptoms.—The main symptoms of gas embolism are those already described under the heading of Pleural Shock—any nervous symptoms may occur according to the area of the brain or cord affected, and they are generally indistinguishable from those which occur as the result of pleural reflex. Possibly the onset is less abrupt in embolism, and the patient may have time to complain of feeling ill before unconsciousness supervenes. Brauer describes the appearance of skin “marbling” where the gas reaches the small peripheral vessels, and says that this is followed by the appearance of pale areas and cyanotic patches, and later by patches of hyperæmia like an exanthem. Characteristic, according to Brauer, is the immediate cessation of respiration and circulation, and he has seen recovery after one minute of such cessation, and after one hour of unconsciousness.

Prophylaxis.—Gas embolism has become a rare complication since the introduction, by Brauer, of the water manometer, and a few simple precautions should render its occurrence well-nigh impossible. These precautions may be briefly enumerated.

Avoid puncture or tearing of the lung. The first is assured by abstention from the use of a sharp needle at the initial operation—the second by extreme gentleness in the use of the blunt needle or cannula, particularly in the presence of adherent pleura. Tearing of the lung tissue may also result from the use of very high intrapleural pressures.

Never open the connection with the gas bottle until, or unless, a characteristic manometer reading with respiratory fluctuations is obtained. This precaution must be as scrupulously followed at refills as at the first operation, since it can never be certain but what the needle has entered a pleural adhesion. Mayer experienced a fatal gas embolism from the withdrawal of the needle during a refill into a tongue of adherent

lung tissue through which it had passed into the pneumothorax cavity.

Let the gas flow in at a decided negative pressure at the first operation by lowering the water-bottle level some 7 or 8 cm. below the level of fluid in the gas bottle.

Avoid any attempt to obtain manometer fluctuations, where these are absent, by getting the patient to take deep breaths or to cough (see, however, p. 68). During unavoidable coughing the tubing should be closed with finger pressure near the needle, partly to cut off the gas, and partly to prevent the manometer fluid being coughed out.

Never pass the blunt stilette for clearing the needle without the use of a stuffing box or a rubber cap. Saugman experienced a fatal accident where this precaution was not adopted.

Do not use rubber and manometer tubing of needlessly large bore, nor have tubing of needless length.

Treatment.—The treatment of gas embolism must be in many respects similar to the treatment of pleural shock (*q.v.*), since it is generally impossible to diagnose between them. The needle must be immediately withdrawn and the patient inverted or the head hung low, since gas tends to rise. Diffusible stimulants—ether, camphor, etc.—must be injected and faradisation of the heart may be tried after the advice of Saugman. Artificial respiration must be strictly avoided, as likely to pump more gas into the vessels, but rhythmical tongue traction may be tried.

SURGICAL EMPHYSEMA

Superficial.—This is apt to occur where a large needle is used, where a high intrapleural pressure is maintained, where the pleura is stiffened by disease, where the patient's tissues are very deficient in tone, and especially where much coughing follows the filling. The manœuvre of lifting and manipulating the tissues leading down to the pleural puncture, so as to obliterate the path of the

needle as this is withdrawn (see p. 47), has practically always sufficed to prevent this accident in the writer's experience. When emphysema occurs a puffy swelling appears, giving the characteristic crackling sensation to finger or stethoscope. A pad and pressure and prevention of cough, so far as this is possible, will generally help to check its further spread.

Deep or Mediastinal.—This occurs where the gas gets under the visceral pleura into the interstitial lung tissue. From there it tracks into the mediastinum and appears in the neck above the sternum. The symptoms are sore throat and a sensation of tightness round the neck, difficulty in swallowing and breathing, and a nasal intonation of voice. Pain in the chest may be present and the patient may become cyanosed, greatly to the alarm of himself and his medical attendant. Nevertheless, no harm ever results, though the condition may recur and even prevent the continuation of treatment.

PERFORATION OF THE LUNG

This, the most serious accident to which a pneumothorax patient is liable, is fortunately of comparatively rare occurrence. Forlanini experienced 8 perforations among 139 patients; all were in the stage of "clinical recovery," before anatomical healing was attained. Jacot found 5 cases, or 20%, among his collection of purulent effusions. The conditions which predispose to this accident are:

(1) The presence of large cavities or caseous areas, and this forms one of the arguments against needless delay when once the patient has passed beyond the reach of other methods of treatment.

(2) The presence of an incomplete pneumothorax in a patient who seeks to live an ordinary life, and this appears to be the case particularly where uncollapsed cavities are present. Saugman experienced this accident in a girl patient of his as the result of riding and dancing.

Those who practise pneumothorax therapy will have experience of patients with large superficial cavities, which separate only partially or with difficulty from the chest wall, and generally maintain firm adhesions elsewhere, and these patients form a constant source of uneasiness on this account. Spontaneous perforation seems to be especially common where advanced disease is present in the lower lobe; wounding of adherent caseous lung at a first operation seems a possible predisposing cause to a subsequent perforation.

Forlanini considers the perforation in all cases due to rupture of a softened caseous area and not to the mere progress of disease—he thinks the risk greatest where the pressure has been allowed to fall too low before a refill. All his cases, save one, were in men, and all considered themselves well and pursued an ordinary life. Jacot particularly insists on the rôle of strong untearable adhesions in the causation of lung perforation; he has commonly found the pleural rupture at the point of attachment of a stretched adhesion. He admits, however, the occasional responsibility of a quiet ulceration. The rupture, he points out, is not necessarily into a cavity.

Symptoms.—The symptoms are sudden and violent pain in the side followed by a hectic temperature with a rise of about 3.5° F. (2° C.), and the rapid appearance of a pleural effusion. The signs may not differ much from those already present. When the perforation remains open an intense whistling sound may accompany the respiratory murmur. A drop of intrapleural pressure and inability to re-establish it by the introduction of gas is evidence of considerable value. Often a plug of tissue makes the opening valvular; the manometer may then show a step-like rise of pressure. In addition, a gas analysis, when this can be undertaken, will show an abnormal percentage of oxygen—proof of an open fistula which was demonstrated by Leconte and Demarquay as long ago as 1863.

Prognosis.—Rupture of the lung is generally of fatal

issue, for whereas in spontaneous pneumothorax the resulting collapse of the lung tends to close the wound and favours healing, in these cases, where the lung is already collapsed so far as this is possible, the rupture does not close but even tends to gape still further. The pleura suffers a constant reinfection and a fatal empyema generally leads to death within a year.

Prophylaxis.—Most important is it that the activity of patients with partial pneumothorax and cavitation be kept within reasonable limits, and this particularly at the time when a fresh refill begins to be required. Restrictions should be put on violent and sudden movements rather than on physical exertion of a quiet continuous nature. Patients with complete collapse of the lung do not run this risk of perforation.

Treatment.—Where cases do not die quickly of acute infection, the pleural exudate accumulates and is coughed out from time to time through the lung fistula, either accidentally or as the result of purposeful changes of position. Heetic fever occurs, the patient grows weaker, and amyloid degeneration is likely to follow. For such cases ordinary rib resection offers no hope, as the lung cannot expand and the cavity cannot close. A hopeful method has, however, been devised by L. Spengler, a method which has indeed led to the recovery of seven among thirteen cases so treated. Its aim is the closure of the cavity by a series of aspirations each followed by a plastic operation. The fluid is drawn off and one or two days later an extrapleural thoracoplasty is done over the lower part of the chest. Two or three weeks later a further aspiration followed by a thorax resection of the upper chest is performed. A third operation may be needed, and in a few cases the cavity is then completely closed. In the remainder the marked reduction in size of the suppurating cavity allows a great improvement in the patient's general health, and after a time a Schede's operation, removal of the parietal pleura and soft parts, assures a further closing down of the cavity. Only three operation deaths occurred among

the thirteen cases as against a mortality of 80% for Schede's operation done at an earlier stage. A further three cases died of tuberculosis after weeks or months, leaving seven recoveries, of which four became fit to return to work.

CHAPTER XIV

GENERAL RESULTS OF PNEUMOTHORAX TREATMENT

IN a disease so protean in its forms, and of such different outlook at various stages and in different individuals as is phthisis, no matter needs more cautious and judicious handling than do the statistical results of any mode of treatment. It may be said with much truth that no individual case of phthisis is strictly comparable to any other case, and this fact is especially brought home to him who sets out to generalise on a collection of phthisis material. Nevertheless, generalisation must be attempted, and some sort of statistical compilation is, in the long run, inevitable, since in this way alone can a large material be brought before the eye. With this in mind the writer has collected such figures as are available from the works of certain experienced operators, and puts them forward for what they are worth. Since each author has his own special method of correlating his results, the figures are not often comparable one with another and each must be taken as it stands. The matter is further complicated by the fact that the number of completed cases of any one operator is not, as yet, large, and that the periods since cessation of treatment vary and in many cases are too short to form a basis for conclusions. It may, indeed, be said that the time for a statistical summing up of the results of pneumothorax treatment has hardly yet arrived. Nevertheless, though the ultimate percentage of complete cures is hardly yet to hand, the enormous volume of

clinical improvement clearly emerges from the available figures, and even if the results were not permanent, yet the prolongation of life, increased comfort and well being, and return of power to work—and this for the most part among cases of advanced disease—entitles the operation of pneumothorax to a very high place among successful therapeutic measures.

Saugman (1913) introduces his results with the following remark :

“If you are to judge of the results of the treatment with artificial pneumothorax, it must at once be understood that this treatment deals with very severely attacked third-stage patients, of whom a great part, indeed most of them, without this treatment would have practically no chances of recovery and healing.” After recording his agreement with Brauer as to the impossibility of dealing statistically with these cases, he offers a “kind of statistics” in the following form :

Thirty-seven of his cases were “quite hopeless” owing to disease in the better lung being of considerable extent (covering more than half the surface), or progressing, or owing to complications which themselves threatened life. In 8 of these he failed to produce a pneumothorax and these have all died, 6 within six months, 1 after one year, and 1 after two years. Of the remaining 29 in whom a pneumothorax was produced, 25 are since dead. In some there was great temporary improvement with loss of fever and relative health for some months, with disappearance of the tubercle bacillus from the sputum. Four are still alive, 2 in a rather hopeless condition, but 2 with a possibility of ultimate recovery.

One hundred cases are classified as suitable for pneumothorax treatment owing to the relatively good condition of the better lung, “though often attacked over nearly half its surface,” but then with less active disease. Among these, followed up for periods varying between $1\frac{3}{4}$ and 6 years, the effects of pneumothorax treatment are compared with that of sanatorium treat-

ment among those (36 in number) in which adhesions prevented the production of a pneumothorax:

	Pneumothorax (64).	No Pneumo- thorax (36).
1. Fit for ordinary or lighter work	32	8
2. Unable to work on account of tubercu- losis	18	12
3. Dead from tuberculosis	12	14
4. Unknown	0	1
5. Dead from acute complications	2	1

Neglecting the last three cases the following percentages are given:

1. Able to work	50%	22.2%
2. Unable to work	28.1%	33.3%
3. Dead from tuberculosis	18.7%	38.8%
4. Freed from tubercle bacilli	50%	8.6%

Among the treated cases 50 are alive, and in 30 of them the treatment is completed. Of these, 13 are cured and 2 free of symptoms, but the time interval is still too short for safety. Five are partly able to work and 10 are still ill. In these last 15 cases treatment was suspended because disease was progressing in the functioning lung, and this sufficed to secure its arrest in several cases.

Saugman's mode of dealing with pneumothorax material is probably the best that can be devised, the groups chosen for contrast being very fairly comparable. Indeed the only obvious criticism which comes to mind is the likelihood that the more advanced and less favourable cases might tend to fall into the second group where universal pleuritic adhesions prevented pneumothorax. and, unless the cases were all at closely similar stages of disease, such criticism must be admitted to be valid.

Begtrup-Hansen (1913) follows a similar classification to that of Saugman, but refrains from presenting his material in any statistical form. He divides up his 100 cases as follows:

In 31 no effective pneumothorax was possible. Among 25 of these followed up later, 10 died (40%), 9 got worse

(36%), 6 improved (24%), but only 3 lost their tubercle bacilli.

In 69 an effective pneumothorax was produced; 24 of these were really unsuitable owing to advanced disease of other organs or of the other lung, and 23 of these died, though many showed a temporary improvement.

Of the remaining 45 cases—all treated for over 1½ years, all with severe disease and tubercle bacilli in the sputum, and 38 with fever—8 were completed and cured and 7 of these have remained so for periods of three months to five years, but 1 got a return of disease in the same lung one year later and had a thoracoplastie operation done, since the pleura was adherent. The duration of treatment in these 8 cases varied from eight months to four years. Fourteen other cases are apparently cured, giving a good result in 22 of the 45, or nearly 50%. A further 17 improved (37·7%), 6 got worse; of all, 10 are now dead (22·2%).

Of Zink's (1913) 110 cases, in 81 effective pneumothorax was achieved; in 35 of these it was complete and in 45 partial, though in most of these adhesions were eventually reduced to long strings and nearly full collapse obtained.

Of 67 cases, the remainder being too recent for inclusion, 25 entirely lost their sputum, and in addition 29 became and remained entirely bacillus-free. Thirteen continued to void tubercle bacilli.

Thirty-one became fully fit for work; in 9 of these the lung has been long re-expanded; 22 go about their business with the lung still collapsed. All these give promise of complete recovery. In the 9 cured cases the duration of treatment was 6 months, 9 months, 12 months, 17 months, 20 months, 23 months, and 25 months in 2 cases. In 6 the cure is expected to be permanent, but in 3 the treatment was very short and the future uncertain, although they appear to be healed.

Sixteen cases have got worse, in 5 of these because treatment was not maintained sufficiently long (½ year, ¾ year in two, 1 year in two), though the first effects were

excellent. Of the remainder of these, in 5 disease spread in the other lung, in 3 in the (? partially) collapsed lung, and 1 died of tuberculous enteritis. The remaining 20 cases have all attained the stage of "clinical recovery" and expect to get well, though in one or two this result is less likely.

L. Spengler (1913) discusses the results in 88 patients, of whom 23 (26%), with treatment completed for periods of $\frac{1}{2}$ to 5 years, are apparently cured; a further 10 (11.3%) have improved, but in 19 (21.6%), mostly with partial and ineffective pneumothorax, the results were negative. Thus he obtained a "positive" result in 69 of the 88 cases (78.33%) and a "negative" result in 19 cases (21.66%).

Among *Keller's* (1912) 40 cases, in 25 a complete pneumothorax was achieved and in the remaining 15 no efficient gas collection could be produced. Among these 15 he records a 60% mortality as against 20% (5 cases) among the 25 pneumothorax cases. His cases were operated on by Brauer's open method.

Brauns notes that among 80 of his cases, 49 (61.2%) were healed, and 20 (23.7%) dead in spite of the pneumothorax. Twelve still remained under treatment.

Carpi (1914) records the following results among 36 cases treated :

Six cases completed for $1\frac{1}{2}$ to 4 years were cured :
all but one of these were bad cavity cases.

Twelve have been treated for 1 to 2 years : all in stage of "clinical healing" and 6 at work.

Two cases treated some weeks with improvement.

Three cases began well but treatment had to be suspended on account of the development of disease in the opposite lung or elsewhere.

Thirteen cases died under treatment, 6 of intestinal tubercle which became worse under pneumothorax treatment, 2 of disease in the other lung, 2 of lung perforation, 2 of empyema, and 1 of tuberculous meningitis.

Thus in 55% a good, in 8·3% a doubtful, and in 37% a negative result was obtained.

A. Pisani (1914) divides his cases into one-sided and double cases.

One-Sided Disease.—Thirty-two cases of which 18 have been fully healed for four or five years, 6 have shown temporary improvement, and 8 have died of complications.

Double-Sided Disease.—Seventy-two cases; in 33 of these a decided temporary improvement lasting in some a long and in others but a short time. In the remaining 39 great improvement occurred and symptoms vanished—but later disease progressed in the opposite lung.

So much for the individual records of European workers. The American figures have been conveniently collected by Sachs, who is thus enabled to tabulate the results of pneumothorax treatment among a very large material. One thousand one hundred and forty-five cases from the records of 24 American observers are analysed, 75 of these having been under his own personal care. "It is apparent," he says, "from the large proportion of far advanced cases, 88·7% (with bilateral involvement present in 77%, cavities in 62%, and serious tuberculous complications in 23%), that artificial pneumothorax is being tried at present in this country chiefly in very advanced cases unimprovable under ordinary sanatorium regimen. This must be taken into consideration in judging the 'immediate' and 'subsequent' results obtained in the 1,145 cases analysed in this paper." The "immediate" results are conveniently set forth in the following table:

Quiescent	119 or 10·8%	} 21·7%
Arrested or apparently arrested	105 or 9·5%	
Cured	16 or 1·4%	
Improved	323 or	29·2%
Failure	162 or 14·6%	} 49·1%
Unimproved	203 or 18·3%	
Dead	180 or 16·2%	

The favourable results, he points out, were mostly, that is in 29·2%, of a "palliative" character—in 21·7% they were "apparently durable." A comparison with the combined results of Zink, Brauer, and Spengler shows the European and American results to be fairly closely in accord:

	American.	European.
Unimproved, failures, deaths .	49·1%	42·9%
Improved	29·2%	31·2%
Quiescent	10·8%	9·8%
Arrested and cured	10·9%	16·1%

Among the "apparently durable" results, 21·7% of the American figures, Sachs finds that not much over one-half stand the test of time, as shown by the figure, 12·3%, obtained for cases remaining well after re-expansion of the lung.

These figures, be it remembered, cover the whole of a very mixed material and one in which, with bilateral involvement in 77% of cases, the indications for pneumothorax treatment appear to have been interpreted in a very liberal spirit. The results are also burdened with cases of "partial" pneumothorax, a condition which varies, according to its volume, from fair utility down to complete uselessness. The "immediate results" showed only 18·2% quiescent and arrested for "partial," as against 47% for "complete" pneumothorax. Again, among 162 "bilateral" cases there were only 24·7% quiescent or arrested and 43% unimproved or dead, while among 91 "unilateral" cases 50·5% were quiescent or arrested and only 12% with no improvement or death. These marked discrepancies show clearly how very different are the results under the best conditions of selection and achievement, from those attained in cases unsuitable from the beginning, or where the production of an efficient pneumothorax has demonstrably failed. In discussing the indications for treatment (p. 12) the writer expressed a hope that with extended knowledge fewer and fewer cases would be allowed to drift into those stages where pneumothorax

treatment is but doubtfully indicated, and adhesions are almost certain to limit its utility. To achieve a sure success the operation must be performed at the suitable moment! Under such circumstances, exemplified to some extent in the selected figures presented above, pneumothorax treatment could lay claim to a success little short of marvellous when the nature of the material (see Indications and Contraindications) was fully taken into account. The striking success of the treatment in comparatively advanced cases, more especially the immediate success, impresses itself very forcibly on all those who practise this treatment, and has led to a chorus of praise with never a dissentient voice.

"All the physicians who have applied artificial pneumothorax on an adequate scale," says R. Burnand, "agree in assigning to it an eminent therapeutic value. We have failed to find such unanimity up to now, in the appreciation of any other antituberculous remedy, and this agreement alone is of a kind to give us encouragement." Burnand's own experience allows him to subscribe to this favourable opinion of a method which "is capable of bettering very decidedly, perhaps of curing patients seriously attacked, where no other system of treatment would have a chance of saving them." It is to the "lost" case of phthisis that pneumothorax treatment offers a chance of life and recovered health, and this, as one of its exponents has justly affirmed, is its "high aim."

BIBLIOGRAPHY

BEGTRUP-HANSEN, TH. (Vejlefjord Sanatorium, Denmark) :
 Pages 121, 125, 130, 168 : " Der künstliche Pneumothorax in der Phthisisbehandlung." (Copenhagen, 1912 ; reviewed in *Internat. Centralblatt für Tuberk.-Forschung*, 1913, vii. 362.)

BRAUER, L. (Hamburg) :

Pages 3, 20, 30, 152, 156, 158, 159, 160 :

" Die Behandlung der einseitigen Lungenphthisis mit künstlichem Pneumothorax (nach Murphy)." (*Aerztl. Verein z. Marburg Dec. 1905, Munch. med. Wochenschr.*, 1906, liii. 337.)

" Weitere klinische und experimentelle Erfahrungen über arterielle Luftembolie." (*Verhandl. des Deutsch. Kongress. f. inn. Mediz.*, 1913, xxx. 347.)

BRAUNS, H. (Hanover) :

Pages 52, 55, 121, 170 : " Meine Erfahrungen mit der Forlaninischen Stiehmethode in der künstlichen Pneumothoraxtherapie." (*Zeitsch. f. Tuberkulose*, 1912, xviii. 549.)

BRUNS, O. (Marburg) :

Pages 8, 95, 104, 109, 110, 114, 117 : " Ueber die praktische Bedeutung der Zirkulationsänderung durch einseitigen Lungenkollaps bei therapeutischen Eingriffen an der Lunge." (*Beiträge z. Klin. der Tuberk.*, 1914, xxix. 253.)

CARLSTRÖM, G. (Göteborg, Sweden) :

Page 117 : " Beitrag zur Frage der Wirkung des künstlichen Pneumothorax auf das Herz und die Zirkulation." (*Beiträge z. Klin. der Tuberk.*, 1912, xxii. 243.)

CARSON, JAMES (Liverpool) :

Pages 1, 6 : *Essays, Physiological and Practical.* (Liverpool, F. B. Wright, 1822.)

CAYLEY, W. (London):

Page 2: "A case of hæmoptysis treated by the induction of pneumothorax so as to collapse the lung." (*Clin. Soc. Trans.*, 1885, xviii. 278.)

CLOETTA, M. (Zürich):

Pages 114, 115: "Beiträge zur Physiologie und Pathologie der Lungencirculation und deren Bedeutung für die intrathorakale Chirurgie." (*Archiv. f. Klinische Chirurgie*, 1912, xeviii, 835.)

DAVIES, MORRISTON (London):

Pages 27, 32, 35:

"A note upon the complete removal of pleural effusions by the regulation of intrathoracic pressure during aspiration. (Oxygen replacement.)" (*Lancet*, 1912, ii. 1774.)

"Recent advances in the surgery of the lung and pleura." (*Brit. J. of Surgery*, 1913, i. 228.)

DAVY, J. (London):

Page 32: "Observations on air found in the pleura in a case of pneumato-thorax, with experiments in the absorption of different kinds of air introduced into the pleura." (*Philosoph. Trans.*, 1823, cxiii. 496.)

DLUSKI, K. (Zakopane Sanatorium):

"Beitrag zum künstlichen Pneumothorax." (*Beiträge z. Klin. der Tuberk.*, 1914, xxxiii. 1.)

EMERSON, C. P. (Johns Hopkins University):

Page 48: "Pneumothorax; a historical, clinical and experimental study." (*Johns Hopkins Hosp. Reports*, 1903, ii. 1-450.)

FAGIUOLI, A. (Berlin):

Page 18: "Versuche einer doppelseitigen Pneumothoraxbehandlung." (*Zeitsch. f. Tuberk.*, 1914, xxii. 547.)

FORLANINI, C. (Pavia):

Pages 2, 3, 17, 20, 23, 27, 30, 55, 62, 74, 93, 95, 96, 98, 100, 110, 115, 117, 122, 135, 143, 147, 151, 152, 155, 159, 162, 163:

"A contribuzione della terapia chirurgica della tisi ablazione del polmone? Pneumotorace artificiale?" (*Gazzetta degli Ospedali*, agosto, settembre, ottobre, novembre, 1882.)

"Primi tentativi di pneumotorace artificiale nella tisi polmonare." (*Gazz. med. di Torino*, 1894, lxv. 381, 401.)

FORLANINI, C. (Pavia)—*contd.*:

“Apparate und Operationstechnik für den künstlichen Pneumothorax.” (*Deutsch. med. Woch.*, 1911, xxxvii. 2313.)

“Die Behandlung der Lungenschwindsucht mit dem künstlichen Pneumothorax.” (*Ergebnisse der inn. Mediz. und Kinderheilk.*, 1912, ix. 621.)

GRAETZ, F. (Marburg):

Pages 21, 109, 118: “Der Einfluss des künstlichen Pneumothorax auf die tuberkulöse Lunge.” (*Beiträge z. Klin. der Tuberk.*, 1908, x. 249.)

HAMMAN, L., and SLOAN, M. F. (Johns Hopkins University):

Page 52: “Induced pneumothorax in the treatment of pulmonary disease.” (*Johns Hopkins Hosp. Bulletin*, 1913, xxiv. 53.)

HOUGHTON, JAMES (London):

Page 1: “Account of a remarkable case of pneumothorax.” (*Dublin J. of Med. and Chem. Science*, 1832, i. 313.)

JACOT, M. (Leysin):

Pages 124, 125, 126, 127, 162, 163: “La pleurésie purulente complication du pneumothorax artificiel.” (*Revue méd. de la Suisse Romande*, 1915, xxxv. 117, 185, 241.)

KÜSS, M. G. (Paris):

Pages 3, 27, 35: “La technique et les résultats immédiats du pneumothorax artificiel dans les formes avancées unilatérales de tuberculose pulmonaire.” (*Bull et Mém. Soc. Méd. des Hôp. de Paris*, 1910, xxx. 3^{me} série, 88.)

MAYER, A. (Berlin):

Pages 106, 121, 122, 127, 128, 129, 130, 132, 140, 141, 149, 158: “Experimentelle und klinische Mitteilungen über die nach Pneumothoraxoperationen auftretenden Pleuraergüsse.” (*Beiträge z. Klin. der Tuberk.*, 1914, xxix. 51.)

v. MURALT, L. (Davos):

Pages 120, 121, 122, 124, 130, 132, 135, 142, 144: “Erfahrungen über Exsudate bei künstlichem Pneumothorax.” (*VII Supplement-Band der Beiträge z. Klin. der Tuberk.*, 1914, 356.)

MURPHY, J. B. (Chicago):

Pages 3, 35: "Surgery of the lung." (*Journ. of the American Med. Assoc.*, 1898, xxxi. 151, 208, 281.)

PARKER, R. W. (London):

Page 145: "Suggestions for the treatment of special cases of empyema by thoracentesis and the simultaneous injection of purified air." (*Lancet*, 1882, i. 689.)

PIPER, H. (Berlin):

Page 157: "Ueber die Beziehungen zwischen venösen Blutdruck und intrathorakalem Druck." (*Arch. f. Anat. und Physiol.*, Phys. Abt. 1913, s. 385.)

POTAIN (Paris):

Pages 2, 145: "Des injections intrapleurales d'air stérilisé dans le traitement des épanchements pleuraux consécutifs au pneumothorax." (*Bull. de l'Académie de Méd.* 1888, 3^{me} série, xix. 537.)

POWELL, R. DOUGLAS (London):

Page 114: "Notes on the Pneumothorax occurring in Phthisis." (*Med. Times and Gaz.*, 1869, i. 112, 166, 194.)

RIST, E. (Paris):

Page 108: "Critical Review, Artificial Pneumothorax." (*Quarterly J. of Med.*, 1913, vi. 259.)

RIST, E., and MAINGOT (Paris):

Page 107: "Examen radiologique et pneumothorax thérapeutique." (*Bull. et Mém. Soc. Méd. des Hôp. de Paris*, 1912, xxxiv, 3^{me} série, 377.)

ROBINSON, S., and FLOYD, C. (Boston):

Pages 3, 47: "Artificial pneumothorax as a treatment of pulmonary tuberculosis." (*Arch. of Int. Med.*, 1912, ix. 452.)

SACHS, T. (Chicago):

Pages 52, 153, 171: "Artificial pneumothorax in the treatment of pulmonary tuberculosis; results obtained by 24 American observers." (*J. of Amer. Med. Assoc.*, 1915, lxxv. 1861.)

SAUGMAN, CHR. (Vejlefjord Sanatorium, Denmark):

Pages 20, 27, 28, 29, 30, 43, 46, 48, 52, 62, 77, 80, 95, 96, 97, 117, 120, 121, 129, 141, 152, 155, 158, 159, 162, 167:

"On the results of the pneumothorax treatment of phthisis." (*Trans. of 17th Internat. Congress of Med. London, 1913, Med. Sectn., vi. 463.*)

"Zur Technik des künstlichen Pneumothorax." (*Beiträge z. Klin. der Tuberk., 1914, xxxi. 571.*)

SPENGLER, L. (Davos):

Pages 17, 144, 164, 170: "Der Ablauf der Lungentuberkulose unter dem Einflusse des künstlichen Pneumothorax." (*Correspondenz-Blatt. f. Schweizer Ärzte, 1909, xxxix. 801.*)

SPENGLER, L. (Davos), and SAUERBRUCH (Zürich):

Pages 123, 124, 142, 143: "Die chirurgische Behandlung der tuberkulösen Pleuraexudate." (*Münch. med. Woch., 1913, ix. 2825.*)

STRAUB, H., and OTTEN, M. (Tübingen):

Page 14: "Einseitige vom hilus ausgehende Lungentuberkulose." (*Beiträge z. Klin. der Tuberk., 1912, xxiv. 283.*)

WEBB, G. B., GILBERT, G. B., JAMES, T. L., and HAVENS, L. C. (Colorado):

Page 32: "Artificial pneumothorax with report of gas analyses and experiments to determine the use of air or nitrogen." (*Arch. of Int. Med., 1914, xiv. 883.*)

WEISS, A. (Görbersdorf):

Pages 17, 20, 23, 52, 79, 117, 138, 147, 149: "Ueber Komplikationen bei der Behandlung mit künstlichem Pneumothorax." (*Beiträge z. Klin. der Tuberk., 1912, xxiv. 333.*)

WILMS (Heidelberg):

Page 62: "Die Pfeilerresektion der Rippen zur Verengerung des Thorax bei Lungentuberkulose." (*Die Therapie der Gegenwart, 1913, liv. 17.*)

ZESAS, D. G.:

Page 135: "Zur Frage der pleurogenen Reflexe." (*Zentralbl. f. Chirurg., 1914, xli. 371.*)

ZINK (Davos) :

Pages 17, 23, 47, 52, 98, 123, 156, 169 : “ 110 Fälle von künstlichem Pneumothorax und die daran gemachten Beobachtungen.” (*Beiträge z. Klin. der Tuberk.*, 1913, xxviii. 155.)

INDEX

	PAGE
Acute forms of tubercle, their suitability for treatment	21, 21
Adherent pleura (see Pleura)	
Adhesions (see Pleural adhesions)	
Altitude, effect of, on pneumothorax	80
Anæsthetics and anodynes	42, 43, 44, 65, 154
Antiseptic: solutions for gas bottles	24, 37
preparation of skin	44
Apical phthisis: its suitability for treatment	16
early phthisis a disqualification	19
Apparatus: general features	24
its preparation for use	25, 37
Pearson and Lillingston's	26
de Carle Woodcock and Lister's	27
Küss'	27
filling the gas bottle	37, 38
Asthma as a contraindication	22
Autoinoculations after fillings	88, 89, 103
Autotoxæmia, its removal by pneumothorax	8, 9
Bacteriology of pleural effusions	129
Bed at beginning of treatment	87
Bilateral pneumothorax	18
,, tuberculosis, as a contraindication	17, 18, 172
Blood pressure in artificial pneumothorax	118
Bottles, "gas" and "pressure"	24
advantage of movable	28, 147
fixed	27, 147
Chemistry of pleural effusions	127
Collapse, complete	71
Complete pneumothorax	56
Contraindications to treatment	11
special	22, 23
Cough, effect of pneumothorax on	90, 103
Cytology of pleural effusions	129
Diabetes, as a contraindication	23
Diaphragm, effect of pneumothorax on	106
paradoxical movements of	107

Heart murmurs	117
embarrassment	117, 118
enlargement	118
Hilum tuberculosis	13, 15
History of pneumothorax treatment	1, 2, 3
 Indications for treatment	11
Intervals between fillings, at early stage	64
at later stages	74
in partial pneumothorax	76
Intestinal tuberculosis a contraindication	23, 170
Intrapleural pressure (see Gas pressure)	
Intravenous pressure and gas embolism	157
 Kidney disease as a contraindication	22
 Laryngeal tuberculosis and pneumothorax	22, 23
Lung circulation	114
Lung, perforation of	127, 162
symptoms	163
prognosis	163
prophylaxis	164
treatment	164
Lung, The compressed	108
re-expansion of	95, 96, 98, 99, 111
foreible re-expansion of	133, 142
fibrosis of	95, 96, 108, 109
unexpanded	100
blood supply	115
Lung, The functioning or better	17, 111
effects of treatment on	17, 111, 112
disease in	17, 112
blood supply	111, 116
 Manometers, water	25, 28
most suitable gauge	29
mercury	30
air pressure	30
safety	29
Manometric readings	45, 47, 66
absence of manometric response	49
incomplete manometric response	50
absence of respiratory fluctuations	50
effect of pleural pockets on	50, 51
abdominal puncture	51
at refills	66
respiratory fluctuations	29, 69
significance of various types of	70
effects of pleurisy on	136, 137, 138

	PAGE
Mediastinum	104
effect of open pneumothorax on	104
effect of closed pneumothorax on	105
hernial protrusions of	106
excessive displacement of	112, 113
Mixed infections, risk of	93
 Needles, for initial operation.	 34
writer's trocar and cannula	34, 45, 149
for refills	35
the Lillingston	36
the Sangman	36
sterilisation and drying	43, 44
 Oedema of functioning lung	 102, 112, 113
Operation, Initial (see also Refills)	
technique of	39
sites for puncture	40
preparation of patient	42
of instruments, etc.	42
position of patient	43
passing the needle	43
entrance of gas	45
removing the needle	47
difficulties and complications	48, 49, 50, 51
 Pain, a symptom of pleurisy	 134
Paravertebral triangles and quadrangle	85, 139
Partial pneumothorax, ineffectual	53
effective	55
Perforation of lung (see Lung)	
Peribronchial tuberculosis	13
Physical signs, general	82
after initial filling	48
of complete collapse	73
in re-expansion of lung	99
in pleurisy	138, 139
Phthisis (see Apical phthisis)	
Pleura, adherent, its diagnosis	41, 60
its incidence	52
its reduction	61
stripping of surfaces	79
Pleural adhesions—causing lung perforation	163
Pleural fistula	149
Pleural pockets (see also Partial pneumothorax)	50, 53
Pleural shock or reflex	151
symptoms	151
conditions of appearance	152
prophylaxis	154
treatment	155

INDEX

185

PAGE

Pleurisy, its effect on gas absorption	76
prevalence	120
causation	121
dry pleurisy	123
pleurisy with effusion	123
classification of effusions	123, 125, 130
purulent effusions	125
chemistry of effusions	127
cytology of effusions	129
bacteriology of effusions	129
effects of pleurisy, beneficial	131
unfavourable	133
symptoms of	134
effects on gas pressure	137, 138
physical signs	138, 139, 140
treatment of	140
chronic effusions	141
acute serous effusions	142
acute purulent effusions	143
Pressures (see Gas pressures)	
Pulmonary relaxation (see also Mediastinum)	112, 113
Radiography (see X rays)	
Refills: operation technique	64
puncture spot	64
manometer readings	66
putting in gas	70
early fillings	71
complete collapse	71
intervals	74
intrapleural pressures	77
Replacement (see Gas replacement)	
Results of pneumothorax treatment	166
Safety syringe of Forlanini	36, 150
Succussion splash	139
Suitable cases for treatment (see Indications)	
their number	11
Symptoms, showing complete collapse	73
disappearance of—under pneumothorax	88
of pleurisy	134
of pleural shock	151
of gas embolism	160
of lung perforation	163
Temperature (see Fever)	
Termination of treatment	97
Tidal percussion in diagnosis of adherent pleura	40, 61, 99
Toxæmia (see Autotoxæmia)	
Treatment of pleural effusions	125, 127
of pleural shock	155

	PAGE
Treatment of gas embolism	161
of lung perforation	164
general results of pneumothorax	166
Tubercle bacilli, disappearance under treatment	92, 168, 169
Tubing, rubber, for pneumothorax apparatus	33, 38
as source of gas embolism	158
 U Unilateral tuberculosis	 12
its identity with hilum tuberculosis	13
its incidence	14
 W Weight, effect of pneumothorax on	 92
 X X-rays, in unilateral tuberculosis	 13
value in initial stages of treatment	39
in diagnosis of adherent pleura	61
complete collapse	72
in pleural effusions	139, 140

